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SCLEROSIS OF THE RETINAL VESSELS

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Continued.

Translucency of the Retinal Vessels

The translucency of the retinal vessels is their finest quality and it usually is the first to disappear when any pathological change occurs in them. The cause is not always sclerosis; any toxaemia, for example, tonsillitis, measles, diphtheria, etc., can change their translucency, and can do so in a short time. Bailliart states that he saw the translucency of the retinal vessels disappear in a few hours. I have had no opportunity of verifying this statement, but I believe it is quite probable. Everyone knows how quickly some infectious diseases attack the myo- and endocardium —why should not the same happen to the retinal vessels? That is why we often see young and healthy people with soft arteries and normal blood pressure in whom the translucency of the retinal vessels is lost. Careful enquiry will, in most cases, elicit a history of some toxaemia in their past; but the salient point, which was the subject of my first paper, is that one may see, although rarely, a person with advanced involutionary arterio-sclerosis and good translucency of the retinal vessels, provided the blood pressure is normal. The appearance of essential hyperpiesis, even in a young

person, means a quick loss of retinal translucency as a first change. For eighteen months I tried to verify my own findings; I think they were substantiated. I found a few cases of people of middle age and even of over 60 years, where the translucency of the retinal vessels was still preserved. On the other hand, I never saw a case of diabetes with translucency present. In all my cases of arterial hypertension, the translucency was lost. That is why I divided my sclerotic cases with loss of translucency into two classes; one, where the blood pressure was normal, the maximum diastolic pressure being 90 and the systolic 150-160—in a few cases only was the systolic 170-180, but then the diastolic was only 85; and two, where the diastolic was 90 and over and the systolic from 150-160 up to nearly 300 in a few cases. In the first group, the average age was 47 years 4 months, the blood pressure 128/78, and of the 169 cases, 77 were males and 92 females. The youngest patient was aged 21 years, and the lowest reading of blood pressure in a patient was 110/50. In the second class, the average age was 52, medium blood pressure 200/110, and of 269 cases, 107 were males and 162 females. The youngest patient was aged 30 years, and the lowest blood pressure in one patient was 180/95 or 130/100. The highest in one patient was 240/175, 280/160, 210/160, 300/140.

A man, aged 65 years, was seen by me over two years ago. He was case No. 13 in my first paper. Bad pyorrhoea, deaf, hard arteries, arterio-sclerosis second degree, blood pressure 130/75. Translucency was present in most vessels, the veins when crossed by arteries were not crushed and the shape was not changed, only white lines accompanied the arteries at the crossing points. Slight tortuosity, if any, of medium arteries and arterioles. Eighteen months after this examination, I found that the translucency of the vessels was nearly gone. Definite white lines were present at the crossings. In the right eye, a branch of the sup. temp. vein was partly crushed by the artery. On April 9, 1927, the translucency was completely gone in both eyes. Nearly all the veins were deflected centrifugally and partly crushed. Blood pressure 115/85. The man was much more senile than previously. On November 5, 1927, the blood pressure was 115/70. Definite arterial pulsation was seen on the left disc, where the arteries formed a kind of irregular rhombus, and partly on the retina. It was not so distinct in the right eye. Tension by palpation was normal. Lens opacities in the right eye.

A man, aged 58 years, was seen by me for the first time over three years ago. It was case No. 12 in my first paper. The blood pressure was 190/90. Translucency was absent in the arteries, but was still present in the veins, especially in the right eye. Light reflex was altered. There was slight tortuosity of the

small vessels. Two years afterwards, his blood pressure rose to 200/95. B.E. translucency lost, copper-wire colour, light reflex dotted. White lines on the disc and crossings. Veins were crushed on the disc. No changes at the retinal crossings. He was hypochondriacal and had severe constipation, but was not debilitated bodily.

Case No. 19 in my first paper—a man aged 54 years, blood pressure 160/90, heavy smoker. No central scotoma for colours, B.E. translucency present, small arteries tortuous. Two years later his blood pressure was 180/90. Translucency absent, copper-wire colour, light reflex dotted. The veins were crushed on the disc. White lines on the disc and crossings. Little change at the crossings. *L.E.* A medium branch of the inf. ret. vein was centripetally deflected by a crossing artery.

These three cases speak for themselves; with advancing years sclerosis sets in; first the translucency goes; immediately afterwards, or practically at the same time, appear the white lines at the crossings—and then arterio-venous compression begins.

Two facts are important. The first is that involutionary arteriosclerosis does not attack retinal (and also cerebral) vessels in the first instance, but only much later, or not at all. Bridgett proved this microscopically by examination of the central artery of elderly and sclerotic people. The second is that essential hyperpiesis will alter the retinal (and therefore certainly the cerebral vessels also) at an early age, quickly and permanently. As far as translucency is concerned, essential hyperpiesis undoubtedly attacks the retinal vessels as a site of predilection from a clinical point of view.

Together with the loss of translucency, the colour of the arteries is usually changed. Instead of normal, rosy ones, they appear " copper-wire." The explanation, of course, lies in the sclerosis of the arterial wall, as the colour of the blood remains the same. However, sometimes one may see a case where the translucency is already lost, but the colour is still normal. In the group with normal blood pressure, except in two cases where the translucency of the vessels was not completely lost, we have translucency absent in 98.75 per cent., the copper-wire colour present only in 79 per cent. In the group with abnormally raised blood pressure, the translucency is lost in 100 per cent. and the copper-wire colour is seen in 95 per cent.—again essential hyperpiesis aggravates the condition.

Together with the copper-wire colour goes the " dotting " of the light reflex. This is a very delicate sign and its nature is still unknown. It cannot be the hyaline spots in the arterial wall, as it is seen too early for them to be present, and they would have to be more numerous. There is a slight discrepancy between the percentages of " dotting " and " copper-wire " colour in the first

group: 76 per cent. and 79 per cent. respectively. One cannot, however, rely on this as a mistake is so easily made. In the second group they are the same. In my opinion, both of them can be of mixed origin, caused by a spasm of the muscularis media, and by histological sclerotic changes in the arterial wall.

White Lines at the Crossings

As mentioned previously, the white lines appear on the retinal crossings immediately after the loss of translucency of the vessels. Only once did I see them in a normal fundus. This origin points to their nature; they are the *sclerotic* walls of the vessels. If the reader will remember the complicated structure of normal retinal vessels, and consider that these lines always appear on the extreme outside of the vessels, he will be inclined to see in them, as I do, sclerosis of the " perivascularis," but not of the whole vessel wall. As the vessels run through channels in the retina, elastic ones to allow of their pulsation, but still fixing them firmly by the membrana basalis gliae to the supporting tissue of the retina, the walls of those channels will be reinforced at the points where they cross one another. Invisible in the normal fundus, they will be the first to be seen when it becomes sclerotic. Accordingly, they are seen in 44 per cent. in the first group, being by far the earliest of the sclerotic changes, and in 66 per cent. in the second group; here again the arterial hypertension aggravates the condition. In diabetes they are seen in 65 per cent. Like loss of translucency of the vessels, " copper-wire " colour and " dotting " of the light reflex, they never disappear once they are established. The pathological process may improve, the blood pressure may decrease as a result of medication or *vis medicatrix naturae*, but they will always remain as a permanent witness of sclerosis. Krückmann also points out in his paper the fact that the neuroglial cells, which form the membrana gliae perivascularis, are most numerous just at the angles where the arteries branch. The width of both perivascular sheaths on either side is, in his preparations, equal to half of the lumen of the vessel itself.

The important fact that these white lines at the crossings represent only the sclerosis of the " perivascularis " will be realised when we describe the white lines accompanying the vessels not at the crossing points, but on the retina itself. The opinion is unanimous that they represent a perivasculitis, an actual oedema and inflammation of the " perivascularis." They are present in syphilis, nephritis, and retinitis of pregnancy. But they look different. They are brilliant white, coarser and rougher than the white lines at the crossings, the latter being fine, only whitish, imperceptibly fading into the wall of the vessel, very

difficult to draw, and in all my drawings they are a little exaggerated. This was not the fault of the excellent worker of Hamblin, but my own, as I wished to emphasise them, as seen by red-free light and in different phases of the pathological process.

White lines on the retina only, I saw very rarely; in 2 per cent. in the first group, 4 per cent. in the second, and 4 per cent. in diabetes. They represent consequently a very small minority, being sclerotic or inflammatory. If a scheme were to be drawn up it would be, according to my cases, as follows:—

(1) Physiological—white lines on the disc. On the disc and the retina in the immediate neighbourhood of the disc usually without involving the crossings, or only those nearest the disc, the white lines being absent on other retinal crossings.

(2) Sclerotic—white lines on the retinal crossings.

(3) White lines on the retina only—always a serious pathological feature.

(4) White lines along the vessels—from the disc to the periphery of the retina—an extreme degree of pathological change.

The reader will find examples of statement No. 2 of the above scheme in illustration Nos. 21, 24, 28, 30, etc.

Statement No. 1, mentioned above, is demonstrated by illustration No. 6; No. 3 by illustration No. 7; and No. 4 by No. 8. The reader must remember that in the living eye all these particulars are finer and more striking.

I think I can prove by a few of my cases that the white lines are really due to sclerosis of the "perivascularis" and that this sclerosis actually presses on the vessel wall. Such is the condition in Fig. No. 28. The reader will see, on the nasal side of the disc, a small arterial branch crossing a small vein. (This was really in the other eye of the same patient and was transferred to this picture for economic reasons.) White lines do not accompany the vessels on the disc, but they go for a distance on both sides of the crossings. When first seen, in an earlier stage, they formed a whitish field, through which the vessels were running. The vein was "banked," not immediately before the crossing as is usually the case, but much farther away, where the white lines disappear. Now, at the time of writing, a few months after the drawing was made, the white lines are coming closer together and the "banking" of the vein is gradually getting nearer to the point of crossing. Is this not evidence of actual pressure of the sclerotic perivascular channel, from without, on the venous wall?

What will happen if a vein passes through between an artery and its branch, crossing through their junction as between the prongs of a fork? Or if it goes through a similar arterial fork, but

reversed downwards? In the normal eye nothing will happen to the vein, but it will be different in a sclerotic eye. It is always changed. The earliest stage is seen in illustration No. 22. The end of the inf. cent. vein goes through and over the branching of the inf. cent. artery; the vein is slightly " banked " before the crossing and slightly crushed by the crossing. White lines accompany the crossing vessel on both sides. In illustration No. 30 is seen a more advanced stage. If in the previous case the vein crosses under the artery and thus may be " banked," now it crosses above the artery. In spite of this the vein is distinctly collapsed, although it passes from above downwards. Also fine white lines are present at this crossing. Another possible explanation, instead of actual compression of the vein by sclerosis of the " perivascularis " at the point of crossing, would be by gravity—the vein makes a curve and simply hangs down because its collapsible wall cannot sustain the weight of the blood; but this explanation is untenable. The formation is due to a congenital pre-existing correlation of the vessels; and in the normal eye the vessels cross one another in most fantastic combinations without any such abnormal change. Moreover, I have seen a similar combination in a normal eye without any alteration.

This is well shown in illustration No. 29. The vein crosses *over* the artery. Observe how grossly it is banked and enormously distended. Fine white lines accompany the crossing. Immediately after the crossing the proximal end is entirely collapsed. It was indeed striking to see it as a fine violet band, running parallel and above the artery to join the sup. temp. vein on the disc. Beautiful white lines, like a white field accompany the vein, lying between it and the artery. How can all this possibly be explained except by actual compression of this particular vein at the point of crossing? Or, by collapse of the vein, which is crossing over a harder artery. But this practically means the same thing—sclerosis of the " perivascularis " of the artery.

Further proof of the sclerotic " perivascularis " is given by the white lines between the vein and the artery. We can immediately exclude perivasculitis; it would be on both sides of the attacked vessel, not only on the side common to both of them. In my opinion, it is simply the result of two channels of interstitial tissue running parallel close one to another. Invisible to us in the normal eye, they will become visible if attacked by sclerosis where their walls are superimposed or simply adjacent to one another and, therefore, naturally, more evident. The collapse of the vein in this case has made the channel more visible, as it was originally formed for the same vein when it was not sclerotic, nor collapsed, *i.e.*, much larger. These are, what I call in my analytical tables, white lines between the vessels. They are best seen by red-free

light. I saw them in 3 per cent. of my cases in the first group, and in 5 per cent. in the second group—here again, according to the rule, essential hyperpiesis aggravates the condition. How can this be explained otherwise than by sclerosis of the " perivascularis " ? The vessels only touch one another; they lie on the same level. This is seen frequently in the normal eye without any change, of course. Therefore, Bailliart is inaccurate in stating that the vessels never run parallel, as this would interfere with vision. It seldom occurs, but it does happen, and they may run parallel to one another for a considerable distance. Sometimes it is difficult to differentiate between artery and vein, so close together do they run. In the sclerotic eye, sometimes only a slight contact of the artery is sufficient to depress the vein and bank it. I even saw this in the process, so to speak, of formation. A sup. temp. artery only just touched the vein, the artery being above. The vein was crushed, nearly collapsed, with actual interference of its circulation, the vein being distended just before the point of contact. *Farther on, the same artery again crossed the same vein twice, without any change of the vein.* Can this be explained in any other way than by localised sclerosis of the " perivascularis " channel, where the artery touches the vein, as signified by the white lines between them? Sclerosis of the " perivascularis " does it by *aggravating the pre-existing condition* with all its mechanical consequences, *but not by creating a new correlation of the vessels.* On plates Nos. 21 and 27, one can see what perivasculitis is like; on the temp. sup. artery it is whitish-blue, and runs on both sides of the vessel. The inf. ret. vein and the artery are running parallel to one another and on the same level. The picture, No. 27, is incorrect in that the vein is distended immediately before and again after the crossing of the artery, and a portion of the vein is " banked " between the crossing and the point where the artery touches the vein and the white lines between them begin to be visible. The description is that the lower ret. vein is only touching the artery and the vein is depressed, crushed and " banked." The foregoing phenomena have, as far as I am aware, not been previously described and, of course, not explained. They have nothing to do with the usual arterio-venous compression at the crossing, as the vessels only touch one another.

Another illustration can be seen in Fig. No. 23. Just before the first branch of the sup. temp. artery crosses the vein, the artery touches the vein; the vein is collapsed and white lines run between the vein and the artery. That this arterial branch is a real impediment to the venous circulation is shown by the *centripetal* deflection of the vein and its " banking " before the crossing. If the artery did not touch the vein, the latter would be, most probably, banked after crossing, as usually happens. In one case the

narrowing and banking of the vein occurred in eleven days. The reader can therefore see how important it is to examine the fundus *frequently and thoroughly*.

To recapitulate: In my opinion, when the vessels cross, their " perivascularis " forms a reinforced channel, which binds them together; the white lines at the crossings are the first sign of sclerosis of those channels, which may actually press on the veins. The white lines between the vessels are the sclerotic " perivascularis."

I did not find much in the literature on this important subject. Either it is simply ignored, or the statements are indefinite and diverse matters are mixed together in one clinical picture. Frost in his atlas (Fig. No. 84) faithfully reproduces the white lines which accompany the sup. temp. vein especially where it crosses the artery, but he does not describe or explain them. In Fig. 87, if I am not mistaken, the periphlebitis of the sup. temp. vein is drawn, but not described. On the other hand, he noticed the white lines and even the fact that they were on the outside of the vessel, p. 47. In de Schweinitz's book, I found only one reference, on p. 519: " These (vasculitis and perivasculitis) are characterised by the appearance of white lines along the vessels, or, rather, the vessel walls become apparent by their conversion into whitish tissue, due probably to an infiltration of the adventitia, composed of lymph-corpuscles." In Gowers' book I found only the one reference, on p. 198, which I have already mentioned. On p. 199, he mentions the rare case where the blood column can still be seen in a minute part of an otherwise opaque vessel wall. He thinks that this is a sign of an anastomosis. It seems to me that the explanation may be simpler; the wall of the vessel has lost its translucency and the perivasculitis does not allow the blood column to be seen, but if a small point of the vessel wall has escaped the perivasculitis, then, of course, the blood column will be seen there. It is a minor point and I do not insist on the correctness of my explanation, but the reader will see later that sclerosis can attack a minute part of the vessel wall, as by predilection, without damaging the whole vessel, and *vice versa*. On p. 210 he states—" It has been thought that a careful recent microscopic examination of the nervous tissues elsewhere reveals the occurrence of similar changes in them. Gull and Sutton state that an extensive increase in the supporting tissue of the nerve centres may be found in chronic Bright's disease and the thickening in the supporting tissue of the retina may be part of this change." In Parsons' Pathology I found a few lines on p. 1286. On p. 1287, he makes an important remark " fibrosis would be a more appropriate term for some of the changes, and they are no doubt to be looked upon as a preservative tissue reaction against abnormal circumstances in the circula-

tion, a strengthening of vessel walls against undue strain." In Fig. 434, he shows fibrosis of the connective tissue in the wall of the vessels, with intact intima. When speaking of perivasculitis, he states that " it appeared that the longer the process went on, the denser became the perivascular connective tissue." In Graefe-Saemisch's Handbuch, Vol. VII, 1st abt., p. 84, I found a description of changes in the vessel wall, caused by chronic inflammation. They consist in hyperplasia of the tissue elements and interstitial connective tissue, *Wucherungen* (? local growth) of the intima and thickening of the adventitia. The proliferation of the interstitial tissue leads to sclerosis and the thickened vessel wall becomes homogeneous and more refracting to light. On p. 92, as criticised above, the white lines on the disc, at the crossings, and on the retina are all mixed up together. On p. 102, it is stated that Rählmann found in 210 cases of arterio-sclerosis (unfortunately no distinction was made as regards arterial hypertension) in nearly 50 per cent. sclerotic vessels—white lines, local narrowing and enlargement. Farther down he describes the views of Gunn and Rählmann on sclerosis of the retinal vessels. In his opinion, there first appears the silver-wire artery, then the translucency of the vessel is (a little) lessened! I found translucency completely lost long before the appearance of the silver-wire artery. My analytical table is the best demonstration of this. Moreover, I am under the impression that Gunn was of the same opinion as myself. In his famous paper before the Ophthalmological Society of the United Kingdom, he states, " Loss of translucency. In health the retinal artery is sufficiently translucent to permit of an underlying vein being detected through it. One of the earliest indications of change is loss of this power of transmitting light, and the difficulty is soon increased by a manifest pressure on the underlying vein." . . . And farther on, " Changes of the coat of the artery . . . are rarely here accompanied by so much fibrous thickening, as to lead to the appearance of the white lines along the vessel." Fuchs mentions only those white lines along the vessels, and, in his opinion, they appear only very late, as I also think: " Conditions of arterio-sclerosis and high vascular tension are often accompanied by visible changes in the retinal vessels . . . in the later stages, white streaks along the arteries and veins (perivasculitis) and haemorrhages. . . . They form a frequent and early sign of general arterio-sclerosis, especially of the brain and kidney." It is necessary to comment on this. First of all, it is not to be thought that conditions of general arterio-sclerosis are often accompanied by distinct retinal changes; they are not. The analytical table shows this; any retinal change, although it may be seen with a normal blood pressure, is still rare compared with the frequency in essential hyperpiesis. Secondly, Fuchs speaks

here of arterio-sclerosis of the brain and kidney—is it atheroma, or senile kidney, or involutionary cerebral arterio-sclerosis, or essential hyperpiesis? In involutionary arterio-sclerosis, therefore, in senile kidney, in my opinion, the retinal changes are not frequent, and, if they are present, they are not early. However, Fuchs' sentence would be entirely correct, in broad terms, if he were to speak not of " arterio-sclerosis," but of " high vascular tension." Gunn, although very vaguely, grasped the difference. " *Old age alone does not produce these changes.* . . . They start between 40 and 50 in predisposed persons and increase more or less gradually thereafter." In my first paper, the average age for moderate hyperpiesis was 51, and for advanced 59—an average of 55. In this paper, the average age for advanced and moderate hyperpiesis is 52. This may be a small point, but I cannot help pausing again and paying tribute to Gunn. I have at my disposal a luminous ophthalmoscope, a sphygmomanometer, and the literature and experience of the profession for over 30 years, while this remarkable investigator, working alone, without any definite knowledge of the medical aspect of arterial hypertension, without modern instruments not only contributed the greatest part of our knowledge, but even correctly pointed out the age when changes usually commence.

I paid special attention to Bailliart's book " La Circulation Rétinienne." It is the only work of modern times which specially describes the retinal vessels and the retinal circulation. A very excellent book indeed, but I cannot agree with many of Bailliart's opinions. For instance, the structure of the retinal vessels is certainly a very complex one, and Bailliart simply mentions that they represent one endothelial tube inside the other. He does not mention at all the important paper of Krückmann, although the names of authorities are very abundant in his book. That is why he makes a definite misstatement on p. 15: " All the retinal vessels are situated in the nerve fibre layer and are in contact with the membrana limitans anterior, the only one which separates them from the corpus vitreum." This can only be true if Krückmann's description is incorrect, and personally I think Krückmann is correct. On the other hand, he makes a very correct observation on p. 30, " In the adult and especially in the aged you cannot see such mobility of the arteries as in a child, as if the arteries were fixed to the underlying papillary tissue." On p. 60, he participates in the common mistake of text-books that the venous pulse may be seen *only* on the papilla. On p. 84 and 113, he states, that " the anatomic capillaries cannot make their lumen larger or narrower, because they do not possess muscles in their walls," an idea which has been shown to be completely wrong by the results of the researches of Dale and other English workers. He does not mention

at all " dotting " of the reflex in sclerotic arteries, a phenomenon, which can be seen only after some training, but which is met with very often indeed. On p. 186, he mixes up, in my opinion, three important, but different, signs—loss of translucency of the arterial walls, perivasculitis, and endarteritis. On p. 189-190 he describes the dilatation of the veins before entering the papilla, as mentioned by me in the normal fundus, and regarded as normal by Frost, but Bailliart does not give any explanation at all, and I am afraid that I am left with the impression that he thinks they are pathological—this would indeed be a great mistake. On p. 191 he mixes up, in my opinion, loss of translucency of the vascular wall with periarteritis—two quite different and separate processes. He does not mention at all the "white lines at the crossings," neither does he mention arterio-venous compression.

Tortuosity

Tortuosity of the vessels is often increased; but it is such an indefinite and elusive factor that in this paper I only considered it as present where I saw the " cork-screw " vessels of de Schweinitz or a very definite tortuosity of a certain vein or artery between otherwise non-tortuous vessels. De Schweinitz was the first to describe these cork-screw vessels, and whoever sees them once will never forget the picture, and will agree that the name is very aptly chosen. On the other hand, the fact that such a striking phenomenon was missed by Gunn, Leber, Frost, and others, is the best illustration of the truth of my introductory words to this paper —that sclerosis of the retinal vessels is still incompletely known and investigated, in contrast to retinitis, to which much more attention has been given. Bailliart, if I am not mistaken, does not mention " cork-screw " vessels in his book.

Swanzy thought that "two of the *earliest* signs of sclerosis of the retinal vessels were: Their cork-screw appearance; silver-wire arteries causing more or less a displacement of the underlying veins where they cross the latter (Gunn's sign)." Fuchs puts the signs in the following order:

" (1) Cork-screw appearance of the arterial twigs, while the arteries from which they spring may be normal.

(2) Flattening of the veins by crossings and, later on, ampulliform dilatation of the veins.

(3) A dull red congestion of the disc.

(4) In the later stages—the white streaks along the vessels." In my cases, I saw definite cork-screw vessels very seldom indeed. Very typical " cork-screw " arteries I saw only once, in a woman aged 48 years, with a blood pressure 235/120. The arteries were copper-wire, translucency was lost, and light reflex dotted. The

veins were crushed by the arteries. But even if we reckon all the cases—I saw tortuosity increased in 0.5 per cent. in the first group, in 9 per cent. in the second, and in 8 per cent. in the diabetic group. Silver-wire arteries were present in 0.5 per cent. in the first class, 16 per cent. in the second, and 7 per cent. in the diabetic group. Deflection of the vein in both directions—centrifugally and centripetally—was seen in 26 per cent. in the first, 50 per cent. in the second, and 60 per cent. in the diabetic group. Am I not therefore justified in saying that the cork-screw appearance of the vessels is neither an early nor a frequent sign? Moreover, this may be the reason why it was missing in the observations of Frost and especially of Gunn.

I should like to mention that it would be incorrect to emphasise, as Fuchs does, the arterial character of the cork-screw vessels. In illustration No. 13 most of the veins are of a cork-screw appearance, and the arteries are not affected at all. I am able to report a noteworthy example. It was a case of a woman aged 50 years, blood pressure 160/90, with an advanced sclerotic fundus. In the right eye, the inf. cent. artery and vein, not far from the disc, are surrounded by a field of white exudation and haemorrhages, the vein already nearly represented by a white line. Farther down, both vessels, although narrow, are still red; evidently the lumen is not completely blocked by the narrowing nearer the disc and some blood is still getting through. But immediately afterwards and right up to the periphery of the retina, both vessels are completely obliterated. They are narrow white lines. Their branches are obliterated, too; but on 2-3 terminal vessels are aneurysms. They are situated, like grapes, each on a minute vessel, and appear to be not true aneurysms, but a conglomeration of distended capillaries, somewhat like a *caput Medusae*. In my opinion, they are distended venules, because higher up their vessel forms a very contracted vein, which joins the inf. cent. vein between the disc and the first narrowing. If they were arterial, they would be collapsed. Their distension is the result of an impediment to the venous circulation.

Another case was that of a woman aged 55 years, blood pressure 300/140, with highly advanced retinal sclerosis, and a retinal haemorrhage in the right eye. In her left eye, near the inf. cent. artery was an area of thrombosis and all small vessels nearby, arteries and veins alike, were cork-screw. In illustration No. 12 such a cork-screw vein will be seen.

What is the cause of the increased tortuosity of the retinal vessels?

As far as the veins are concerned, there is little doubt that their tortuosity is increased by some obstruction to the outflow of blood. If, as in my case above, the obstruction is at the end of a branch,

the venules will be tortuous. If, in the lamina cribrosa, the large trunks also will be tortuous. It is not so easy to account for the tortuosity of the arteries, and Gunn's explanation may be the correct one: " The tortuosity may be regarded as the result of the *vis a tergo*—the blood current going through a narrowed tube." Sclerotic temporal arteries are also often seen to be unusually tortuous. Sometimes, of course, there may be an obstruction to the arterial circulation, when the artery may become tortuous; but this is rather an exception and not a rule, and is contrary to what Bailliart describes as the usual cause, on p. 182, without mentioning in this connection the name of de Schweinitz.

I have had the opportunity of seeing the tortuosity of the veins increase all over the retina in a case of severe bronchial asthma. This was a man aged 38 years, very stout, with a fatty and diseased myocardium. He nearly died last year from pulmonary infarct. When he is nearly choked by an asthmatic attack, and is very cyanosed, with the conjunctival veins enlarged and blue, his retinal veins are very prominent, tortuous, large and practically black. The discs are clear, and of a deep rosy colour. The arteries are squeezed into the veins, especially the smaller ones, like a rivulet in a gorge, but the arteries are uncovered on either side. All the veins bank, like sausages. When the attack is over, they again become normal. This proves once more that at the crossing points the vessels are fixed to one another and to the retina also. The " banking " of the vein is not produced here by a sclerotic artery, but simply by a narrowing of the veins at the crossing point by connecting " perivascularis." It disappears when the outflow of the blood is again free. A good illustration of a similar condition is Fig. No. 74 in Frost's atlas, where the grossly distended leukaemic veins are cut through by the arteries.

The waves of a cork-screw vessel may be directed in a horizontal or a sagittal meridian.

General Narrowing of the Arteries

It is a general characteristic of sclerotic arteries throughout the body, that they become narrow and elongated. There are evidently two factors at play—the cicatrisation, so to speak, of the arterial wall and the spastic contraction of the muscularis media. But again, as with tortuosity of the vessels, it is very difficult to decide how great, pathologically, is the narrowing. Therefore, I put in my statistics as " narrowing of the vessels " only those cases where the disproportion between the arteries and the veins was very marked. I saw this in 3 per cent. of the first group, 6 per cent. of the second, and 4 per cent. of the diabetic group. Gunn says that the arteries in a sclerotic eye are abnormally narrow

and show an irregularity in their breadth. I divide these signs, as, in my opinion, they have nothing in common. I call the irregularity in the breadth of the vessels "change in the lumen of the vessels," and this will be described presently. I should be very reluctant to designate arteries very often as "narrow." Possibly a partial explanation of the cork-screw arteries may be found in this tendency of sclerotic arteries to elongate and narrow. As there is not much room for the arterioles, fixed by their "perivascularis," they attain the same result by becoming tortuous.

Silver-Wire Artery

This is, as everybody agrees, a very important sign of far advanced sclerosis. The following is the original description of Gunn: "The arteries have an exceptionally bright reflex; the central light streak is very distinct and sharp, while the whole surface of the vessel is of a somewhat lighter colour than usual. . . . *It was seen in many cases with high arterial tension.* . . . Ophthalmoscopic appearance is due to hyaline degeneration of the arterial wall." I should like to correct this description. When working with a powerful ophthalmoscope, like Woolfe's, by increasing the beam of light, I often obtained a silver-wire appearance in the copper-wire artery. The bright reflex in the centre of the vessel becomes more brilliant, larger, and covers nearly the whole vessel; but the difference between the latter and true silver-wire artery will be that fine red lines will be seen on either side of the whitish line. In a true silver-wire artery the whole vessel will be homogeneous in appearance and the red lines will not exist at the sides. Of course, this can be seen and verified only by the luminous ophthalmoscope which did not exist in Gunn's time. It is simply the result of the higher refracting power of the arterial wall and is usually seen on the large vessels near the disc. In illustration No. 15, the reader can see what I mean. The vessels go over a ridge in the retina and meet the light of the ophthalmoscope at a different angle than at other parts of the retina; they have a very strong silver-wire reflex, but are still accompanied by the red lines. Limited by such a description, the silver-wire artery is, comparatively, not so frequently seen. It was observed in 0.5 per cent. of the first group, 16 per cent. of the second, and 7 per cent. of the diabetic group. I say comparatively, because in the mind of Gunn and even some modern writers, silver-wire arteries are associated with arterio-venous compression (Gunn's sign); and arterio-venous compression of any kind was seen in 26 per cent., 50 per cent., and 60 per cent. respectively of the above groups.

In a silver-wire artery it is rare that any part of the vessel is left intact or copper-coloured. Usually, the whole length is involved.

I was often struck by the silver-wire appearance and tortuosity of the small independent arteries around the disc. The change appears in them first, and may be limited for a long time, whilst the other arteries, especially the larger ones, are of copper-wire colour. I am inclined to think that the appearance of those silver-wire arteries around the disc is of much graver prognosis than that of the silver-wire retinal arteries and their branches.

What is the cause of the silver-wire artery? Gunn thought that it was due to the hyaline degeneration of the vessel wall. Leber, in Graefe-Saemisch Handbuch, p. 871, thinks that the silver-wire arteries of Gunn, which may be seen in cases of high arterial tension and also in nephritis, are merely contracted arteries, without an actual histological change. Frost does not mention the name of "silver-wire artery," but he states "that in chronic Bright's disease the thickening is in the outer coat, so that the lumen is not affected. . . . Careful examination, however, will often reveal the presence of this hyaline thickening at the point where one vessel crosses another." In my opinion, the true silver-wire artery is a combination of spastic contraction of the muscularis media, sclerosis of the perivascularis and actual histological degeneration. That in all cases of arterial hypertension we have first of all to deal with a long-standing spastic contraction of the muscularis, which afterwards leads to myohypertrophy, and only later to fibrosis, was shown by me, when discussing the general problem of involutionary arterio-sclerosis and essential hyperpiesis earlier in this paper. Moreover, I tried to show, when describing the white lines at the crossings, that they were due to sclerosis of the perivascularis. We have all the more reason therefore to suppose this sclerosis to be present in the silver-wire artery, which not only is accompanied by the white lines at the crossings, but usually causes in addition a compression of the veins, in which process the sclerosis of the perivascularis also plays a very important part. Leber points out, on p. 84, that in chronic inflammation of the vessels we see a hyperplasia of the interstitial connective tissue and thickening of the adventitia, which lead to sclerosis, with the result that the thickened vessel wall is more homogeneous and more refracting to light. Moreover, the seat of the light reflex is most probably in the perivascularis and the muscularis media. With the decrease of the muscularis media, the silver-wire reflex also diminishes in the secondary and tertiary branches, to disappear altogether in the arterioles. A diseased muscularis, in the early stage of the affection, is most probably contracted, but later on, the elastic and connective tissue elements multiply and replace or rarefy the muscular fibres. The result will be a true histological change with increase of the silver-wire reflex, especially in its width, because all pathological changes where connective tissue is

concerned, be it perivascularis or adventitia, call them white lines or perivasculitis, are characterised by their more or less brilliant and whitish appearance. The third factor, hyaline degeneration, is usually present in far-advanced arterial degeneration throughout the body, and, therefore, in the silver-wire artery also. I am prepared to go farther than that and say that I do not see any reason why a spastic contraction of the muscularis media, pure and simple, should make an artery silver-wire. I do not think that it can seriously interfere even with the translucency of the arterial wall, or play a part in producing the colour of copper-wire arteries. The chief rôle is played by the actual, although slight, sclerosis of the perivascularis and the vessel wall, and not by spasm of its muscular fibres.

How long will it take for an artery to change from a copper- to a silver-wire? In the case of a lady aged 63 years, with constant arterial hypertension of 190-220/115, which seldom went down to 160-175/100 in spite of dieting and medication, the sclerotic fundus was seen by me on August 8, 1926, and the first time that some arteries appeared as silver-wire was on December 27, 1927, more than a year afterwards. It would be difficult to generalise from one observation, but it is important to note that change of colour of the artery is a lengthy process, in contrast to loss of translucency of the arterial wall which can occur very quickly indeed.

Change in the Lumen of the Vessels

This is a very important and grave sign of advanced sclerosis. It consists of widening and narrowing of different portions of the same vessel. An extreme degree of such an affection is the noteworthy case of Rählmann, quoted and reproduced in Graefe-Saemisch, and a similar one on p. 187 of Bailliart's book. When the larger arteries are attacked, they show a simple narrowing, but their colour may still be copper-wire. When the small independent arteries near the disc are attacked, they present a "beaded" appearance and are very often of silver-wire colour. It seemed to me that in many of them, with red-free light, I could see their external contour still unchanged, *i.e.*, there was no actual narrowing from the outside, but only from inside, as if the intima were encroaching on the lumen of the arteriole.

Two remarks are necessary. When describing the normal fundus, I mentioned the antero-posterior waves of the vessels. Such a wave may give the impression of a narrowed portion of the vessel, especially as it is often accompanied by a brighter light reflex; the change of refraction will show the vessel to be of the same contour, but only lying deeper in the retina. Also, the student must bear in mind, that the narrowed portion of the vessel may be so

small and limited, as to measure only a few millimetres and is, therefore, apt to be missed, unless it be specially looked for. Such is the case in illustration No. 21. The nasal branch of the sup. temp. artery is crossing and centripetally deflecting a secondary venous branch; the portion of the artery between the border of the disc and the point of crossing is narrowed.

In illustration No. 35 is seen a change in the lumen of a venous branch of the inf. cent. vein and in the sup. temp. vein and its two branches. In No. 24 a change is seen in the lumen of a small independent ciliary artery down and in, and in No. 21 in the second branch (on the disc) of the sup. ret. artery. What is the cause of these changes? First of all we have to exclude perivasculitis which usually extends along the retinal vessels for a considerable distance and may obliterate them, but, it does not cause narrowing or a " beaded " appearance. This is demonstrated in illustrations No. 7 and No. 8. The generally accepted explanation, based chiefly on the work of Hertel and Rählmann in Germany, and Coats in this country, is that it is caused by a hyperplasia of the endothelium of the intima, endarteritis obliterans. Foster Moore quotes Coats that " the above appearances are due to a proliferation of endothelium with little or no ground-substance between them; it is characteristic for this form of disease to encroach on the lumen in this eccentric manner." Parsons, p. 588, says that " arterio-sclerosis is characterised in the retinal vessels by proliferation of the endothelium of the intima and new formation of connective tissue, especially elastic fibres." Farther on he states: " In very advanced cases is present a widespread endovasculitis obliterans and proliferation of the adventitia, so that the walls are enormously thickened." According to Graefe-Saemisch, Rählmann found in arterio-sclerosis (210 cases) in 50 per cent. white lines and local narrowing; in 22 per cent., only local narrowing. Lurje frequently found a hyperplasia of the endothelium in sclerotic vessels. Bailliart gives a concise extract of the views of Hertel. " Hertel examined the retinal vessels of 14 persons, who, suffering from angio-sclerosis, did not present any appreciable visual trouble, but had more or less clear ophthalmoscopic signs; tortuous course, irregularity of calibre, narrowing of the arteries, accompanied by white lines, slight atrophy of the optic disc. He divides the results of his investigations into two groups. In the first one, there is no localisation of the lesions, which are diffused over the whole length of the vessel. He states that the endarterium is normal, the tunica externa is very rich in connective tissue, and that the elastic fibres are present in great numbers, especially in the tunica interna and media. Hertel at first attached great importance to this development of the elastic tissue, but investigations on the vessels of young and healthy subjects showed him

that this process was in no way characteristic of angio-sclerosis and began in childhood."

In the second group, the more interesting, lesions are localised in spots. There exists a considerable thickening of the endarterium, the tunica interna being six or eight times thicker than normal. The result is a considerable narrowing of the lumen of the vessel, which decreases from 172-180 μ to 92 μ.

Rählmann observes that the vascular narrowings, seen with the ophthalmoscope, depend on the hyaline proliferation of the " mesarterium," noted already by Streiff. The whitish opacities are produced by the thickening and opacification of the " periarterium."

I should agree entirely with all of this as far as the " beaded " arteries are concerned, but how can proliferation of the endothelium alone explain the narrowing of moderate degree of the arteries, where one cannot see any encroachment at all on the lumen, where both contours, the internal and external, are quite clear and straight? By collapse of the arterial wall? But would it collapse if there still be a wide column of blood to prevent it? I was puzzled for an explanation, when what seemed to me a solution presented itself.

It was case No. 35. The changes were seen in the sup. temp. vein and its two branches. The man was aged 59 years, with a blood pressure 200/100. Five years ago he had a paralytic stroke with involvement of the right side of his body; a few months ago the same thing happened again on the right side. In both instances he improved quickly. In the right eye I noticed that the sup. temp. vein was changed in its volume and was covered by a veil (a focus of exudation?). The veil covered the vein completely, but the vein could still be seen through it, although very dimly. This was on January 17, 1927. A few days afterwards, the same veil appeared at the base of a branch of the vein. On February 28, 1927, I made a note in my book: " Both the vein itself and its branch are narrowed at their base; it is now not a focus of exudation, but a distinct narrowing." On March 14, 1927, the focus of exudation around the main vein was disappearing, so that the medial (nasal) side of the vein was clear, but there was a white area between the vein and the artery, running along the vein. The latter was still narrowed. On March 21, a fresh focus of exudation appeared on the vein, which was passing close above the disc. On April 13, the white exudation disappeared everywhere, leaving only narrowing of the vessels, and whitish lines along them. On September 26, I noted that the veins, where the exudation was, were now of more silvery-wire colour, but not the true silver-wire. The illustration shows approximately the state of affairs for the main vein and its upper branch on March

14, 1927, and for its inferior branch on April 19. I was lucky enough to see the same process in a few other cases. In one of them it took 23 days for this veil on the artery to disappear, leaving a local narrowing of the vessel. I was able to demonstrate a similar striking case on the sup. temp. artery to Mr. A. H. Levy at the London Jewish Hospital. By what is this process caused? A general perivasculitis we can discard immediately, only a localised portion of the vessel being affected. Therefore, it must be a localised inflammation of the " perivascularis," a ring-like sclerosis, pressing on the vessel from the outside. That it is the " perivascularis " and not the intima or media is evident from the character of the veil, covering the vessel from the outside. Gradually this mist clears up from the centre outwards allowing the borders of the vessels to be distinctly seen, but still remaining on their sides as white lines. The deepening of the colour of the affected vessel in a few months' time to nearly " silver-wire " is a sign that sclerosis is still going on; and that it is " perivascularis " is again confirmed by the fact that the exudation first disappears from the medial side of the vein and forms a white area between the vessels with narrowing of the vein. It is analogous to the white lines between the vessels, as described and discussed in the chapter on " White Lines." The process appears somewhat different with an artery. It is seen to pass through a focus of exudation which envelopes it, but, in my experience, is not so marked, nor so thick over it, as in the case of a vein. I think the artery is simply seen better through the veil because of its higher refracting power. A few weeks later the artery is permanently narrowed.

When I realised what was going on under my eyes, I was rather puzzled. Was it possible for the " perivascularis " to be sclerosed on one minute spot of the vessel, without attacking a more considerable part? I was greatly relieved when, in the " Annales d'Oculistique " for 1926, p. 802, I found a histological examination of a case of retinitis circinata by Morax, where he found such a condition actually existed. " We think it is interesting to point out the comparatively localised (*circonscrit*) character of the vascular change. . . . We have to deal with a partial sclerosis, affecting certain parts of an artery and not the whole arterial branch."

To recapitulate. Besides the universally recognised local narrowing of vessels from the inside, by endothelial hyperplasia, it may be caused from outside by local sclerosis of the " perivascularis." True enough, I never saw it involve a considerable extent of a vessel, but, theoretically, this is quite possible and, of course, a succession of such local narrowings will cause a change in the lumen of the vessel on a larger scale. The white lines on

the borders of the vessels usually disappear in time—in contrast to the white lines at the crossings, which are permanent. The difference between this process and ordinary perivasculitis is chiefly in direction and length, the former being transverse to the vessel and short, the latter extending along the vessel for a very long distance. We shall now try to explain the famous case of Rählmann, which, if Prof. Leber's statement, p. 96, is correct, the former did not sufficiently elucidate. A woman who suffered for years from chronic parenchymatous nephritis, cardiac enlargement and sclerosis of the larger arteries, had, soon after her confinement, a severe metrorrhagia and suddenly lost her eyesight. Two months after her sudden blindness Rählmann saw her. Both eyes were blind when the fundi were examined. The arteries were extremely narrowed at some places, but wide at others. The vessels did not show any *"tragung"* ("haziness," "loss of translucency "). A few weeks afterwards the patient died and Rählmann examined the retinal vessels histologically. He found only a hyperplasia of the endothelium and explained the whole occurrence as due to an embolism. Leber does not think that this case is a typical one of bilateral embolism, but does not offer any other explanation. The ophthalmoscopic picture may be explained, in my opinion, differently. If the narrowing of the arteries would indicate a blockage by endothelium—embolism, or, more correctly, thrombosis—the arteries would be occluded and obliterated. The widening of the arteries *after* the narrowing, is the best proof that there was still present a blood-stream sufficiently strong to keep the vessels dilated. To this must be added the fact that ophthalmoscopically the vessels were not much changed. Personally, I think that the translucency of the walls ought to have been lost, but Rählmann did not have a luminous ophthalmoscope and therefore could not increase the intensity of the beam of light. If the endothelium blocked the arteries sufficiently, the blood-stream would also be seen interrupted, as is usually the case in typical " beaded " arteries. On the other hand, if the narrowing were from the outside, as in my cases, it would fit in much better. It usually does not greatly change the ophthalmoscopic appearance of the vessels, and, attacking only localised parts of the vessels, leaves the rest as wide as before; also it does not as a rule completely interrupt the blood-stream. The patient was ill for years previously and had ample time to develop this perivascular narrowing of her arteries. The endothelium, from the double provocation of the toxins and of the blood streaming through a narrowed tube, would, of course, proliferate. What I wish to point out is that the picture that Rählmann saw had nothing to do with the sudden and complete blindness. The latter may be explained more easily by the sudden loss of blood, or perhaps by

the thrombi which formed at all the narrowed places. In time, the flow of blood would recommence and the vessels could be seen again, but the blindness would remain. A similar case of mine is described in the chapter on atheroma, but the normal " appearance of the vessels" disappeared in time. In the case of Rählmann the patient died shortly afterwards.

In conclusion, I should like to mention that I saw a congenital change of an artery near the periphery of the retina in the early stage of a sclerotic fundus, with a normal blood pressure. The affected portion of the artery looked like a cirsoid aneurysm. None of the other vessels and their crossings showed any change at all.

The Arterial Pulse

The usual teaching is that the arterial pulse is commonly absent in a normal person and can only be seen in pathological conditions. Of all causes the first may be valvular disease of the heart, especially aortic regurgitation. Vossius thought that it was possible to discriminate in this way between congenital and acquired disease of the heart, but his view has not been upheld by others, so far as I am aware. In text-books is usually found the statement that the arterial pulse may be seen *only* on the disc, and Bailliart also says this. In many of my cases where the blood pressure was normal, in young persons, with marked, although well compensated, valvular disease of the heart, I could clearly see the arterial pulse far away from the disc. A curiosity I saw in one case was a branch of the inf. cent. vein trembling synchronously with the arterial pulsation; evidently the movement was transmitted from an artery touching the branch. Sometimes one can see pulsation, not directly, but by means of the curious light reflex around the vessels, mentioned when describing the normal fundus. In one case, that of a girl aged 21 years, with mitral and possibly aortic regurgitation, well compensated, blood pressure 140/80, the arterial pulse could be seen 2-3 discs away from the papilla, and by more careful examination I could see it even in the secondary arterial branches, 6-8 discs away from the papilla, especially in their curves. The explanation is simple; because of the incompetence of the valves, the heart beats more strongly, and each pulsation means a brisk disturbance of the blood. The arterial retinal pulse in a normal person can be more easily seen if the arteries form waves.

In five sclerotic cases, in the aged, I saw an arterial pulse appear without any lesion of the cardiac valves and with normal tension of the eye-ball, as tested by palpation. In some of them the blood pressure was normal, in others raised. Sometimes the arterial pulse was more marked in one eye than in the other. It was

usually more marked in the eye where the arteries described a curve on the disc. This was the condition in the case fully described in the chapter on translucency in the sclerotic fundus. Both arteries formed a loop on the left disc, and with each pulsation I saw them tremble and elongate as if trying to advance. The movement was distinctly seen on the disc and less clearly on the retina. In the right eye, where the arteries ran without any curves, the pulse was less distinct. His blood pressure was 115-120/75 and he was aged 67 years.

The other case was that of a woman aged 58 years, with a blood pressure of 120/70. I saw her many times and examined her fundi. On August 7, 1927, I described her fundus; translucency lost, copper-wire colour, light reflex dotted. Spots of arterio-sclerosis in the retina. In the right eye, a narrow, but very long, vein was deflected centripetally by an artery. She again came to see me on January 4, 1928, telling me that she felt sick and giddy. Her blood pressure was 140/90. The veins were more crushed by the arteries. The arterial pulse was seen in both eyes, especially in the left one, where the artery formed a sharp curve. It trembled and elongated with each pulsation. I was able to demonstrate this to Dr. Batty Shaw, who saw the case in consultation.

The third case was that of a woman aged 66 years, with a blood pressure of 230/110. On February 15, 1927, I described her fundi: translucency lost, copper-wire colour, light reflex dotted, white lines on the crossings. The veins (most of them) were deflected centrifugally by the arteries, crushed, with interference of the blood stream. In the right eye, the sup. temp. vein was crushed by the first crossing, but was not changed at all by the second one, 2-3 D.D. farther on. I saw her many times afterwards. On January 17, 1928, I noticed a change in the lumen of some veins, especially in the right eye. On January 25, I observed that the tortuosity of the veins was much increased; the arterial pulse could be seen in both eyes, on the disc, especially in the right one, where the artery formed a long arch.

In the first of the above cases, I saw the patient on April 3, 1927, then not till November 5, 1927, when I first noticed the arterial pulse. In the second case, the dates were August 7, 1927, and January 4, 1928. In the third case—eight days. Possibly, in the first two cases, the actual change also took only a short time, but to affirm or deny this would merely be guess-work.

The youngest of my cases was a man aged 43 years, blood pressure 130/85; fundi: translucency lost, white lines on the disc. Otherwise normal. Arterial pulse could be seen far away from the disc. T.n.

The usual explanation of raised intraocular tension or lowered blood pressure, it seems to me, does not meet these cases. My

explanation would be the following. In a vascular system that has not lost its elasticity, the sudden rise of the pulse-wave by the systole is counteracted by the contraction of the peripheral arteries, " peripheral heart." That is why, besides many other reasons, the pulse wave, when reaching the retinal vessels, is so modified, that sometimes it cannot be seen. The tortuosity of the vessels on their way from the heart to the eye decides the issue. Suppose now that the elasticity and the contracting power of the peripheral arteries were lost to a considerable degree and they were simply rigid tubes, every systolic wave would then reach the eye suddenly and forcibly, and it would make the vessels on the disc tremble and elongate, affecting them at their curves where the pulse-wave would strike and change its direction. In other words, I am inclined to think that in my cases the appearance of an arterial pulse simply indicated a high degree of sclerosis, although in three of them the blood pressure was normal.

Arterio-Venous Compression

This, in my opinion, is the most fascinating, the most easily seen, and the most difficult to explain of all the phenomena of retinal vascular sclerosis. Here again, the reader will have a striking illustration of how deficient is our knowledge of retinal vascular sclerosis, its aetiology, significance and mechanism. Gunn was the first to notice and describe it, and gave us most of our knowledge. Others only followed his lead, whilst Prof. Leber devotes only half a page to a description of this phenomenon, chiefly a very brief account of Gunn's and Rählmann's views. The fact that one cannot see the vein in such cases, Leber tries to explain by its being depressed by the artery, and the observer simply does not accommodate for it. This is rather strange. One can accommodate for a long time and also change one's refraction many times, and still not be able to see the vein. " In any case," he writes farther on, " we must not consider as firmly established, that the previously described pressure of the artery on the vein may cause any considerable impediment." If I correctly understand him, Foster Moore in his "Medical Ophthalmology" nearly agrees with the last sentence of Leber: " I am convinced that any appreciable distension of the peripheral portions of the veins in these circumstances, a condition, to which the term of " banking " is sometimes applied, is of quite unusual occurrence." It is most surprising not to find any mention of arterio-venous compression in Bailliart's excellent book; it seems as if this phenomenon does not exist for the learned French writer.

Even the description of this phenomenon by those writers who have paid attention to it, beginning with Gunn himself, is very

short and does not embrace all the variations; and the existing explanation does not correspond with what I saw in my cases. In other words, my facts do not fit in with existing theories.

Gunn states: "In some cases the vein is indistinguishable just at the spot where it is crossed; it is distended peripherally for some distance from that spot. . . . Influence of the arterial pressure on the venous blood stream. . . . The arteries are bright and show marked pressure on the veins." Swanzy wrote: "The artery's rigidity is shown by the indentation of the veins where it may pass over them . . . . The constricted portion of the vessel may be so limited as to involve only a couple of millimetres of its length and is then very liable to be overlooked. . . . The degree of pressure exerted by the artery on the vein may be so slight as merely to flatten the vein or push it aside in the direction of the blood current in the artery, whilst at other times it may be sufficient to constrict the underlying vein, which then appears "banked" or, again, an engorgement of the vein on the distal side of the artery may show that the circulation in the vein is decidedly impeded." Maitland Ramsay when describing the degenerative form of albuminuric retinitis, says, "The artery loses its natural translucency and becomes hard and rigid; and, consequently, when it crosses a vein, it not only conceals the latter from view, but also mechanically exerts so much pressure upon its walls that the blood-flow is hindered." de Schweinitz, p. 519, states: "The last (impediment of the venous circulation by an artery) is a sign of the utmost importance. . . . If the walls of the artery are thickened by disease, it presses upon the vein, pushes it aside, or directly contracts its calibre so that beyond the point of crossing there is an ampulliform dilatation." Gowers, p. 10, says: "Occasionally some of the retinal venous branches are found to be distended in consequence of local pressure, from an overlying artery, whose walls have undergone degenerative changes." Finally, Foster Moore, p. 51, states that the vein is first hidden from view and then follows the deflection of the vein. "The effect attained is that the vein instead of taking a long oblique course under the artery, traverses from one side to the other by the shortest possible route; a similar phenomenon may be seen when a vein crosses over a sclerosed artery." Now, how can pressure by the artery explain the narrowing and deflection of the vein, if the latter crosses *over the artery*? All observers, excepting Foster Moore, missed this occurrence, and he describes it, but does not explain it. Or why, when the same vessels cross one another many times, do they show different changes at different crossings?—as seen in illustration No. 20, where, at the first crossing, the sup. temp. vein lies on top of the artery and in spite of this is narrowed; at the second the artery crushes and banks the vein; and at the third, it deflects the vein

centripetally (against its own direction). At the fourth crossing the vein shows an " hour-glass " contraction, and at the fifth and sixth, it is very little changed. The most important point is, how can the artery which " pushes aside the vein in the direction of the blood current in the artery " deflect the vein *centripetally*—against this direction? These are the questions to be answered and explained for the full understanding and appreciation of arterio-venous compression. Let us first turn to the analytical table.

The first sign, of course, is the impossibility of seeing the vein, simply because the artery has lost its translucency and not because the observer does not accommodate for it. The white lines at the crossings appear next, or simultaneously. They are, in my opinion, the early sclerotic " perivascularis." Then the vein becomes depressed, and later on depressed and deflected. In the first group of my cases, translucency was lost in 98.75 per cent., white lines at the crossings were seen in 44 per cent., veins were depressed only in 7 per cent., depressed and deflected in 6 per cent., crushed and deflected centrifugally in 17 per cent., centripetally in 9 per cent. In the second group the corresponding figures were 100 per cent., 66 per cent., 7 per cent., 6 per cent., 36 per cent., and 14 per cent.; and in the diabetic group 100 per cent., 65 per cent., 7 per cent., 4 per cent., 40 per cent., and 20 per cent. The veins were crushed without interference with their circulation in 14 per cent., 18 per cent., and 35 per cent. respectively; with interference in 12 per cent., 40 per cent., and 20 per cent. These statistics will help us to clear up immediately a few important points.

According to de Schweinitz, Wagener thinks that " in consecutive arterio-sclerosis, due to general vascular hypertension, there is a *straight arterio-venous compression* (italics of de Schweinitz), *i.e.*, the course of the vein is not altered." It was altered (in both directions) in 26 per cent. in the first group of my cases with normal blood pressure, and in 50 per cent. in the second, where the blood pressure was raised. Depression, and depression with deflection, of the veins are remarkably constant in all three groups. This is because, in my opinion, they represent an earlier stage; they soon change to more advanced signs of arterio-venous compression. I simply interpret this as due to the fact that I saw such a proportion of the earlier stage in all my cases. On the other hand, if we take all forms of arterio-venous compression together—the figures will be 39 per cent. in the first group, 63 per cent. in the second, and 71 per cent. in the diabetic group. The corresponding figures for the white lines will be 44 per cent., 66 per cent., and 65 per cent. This means that in the first and second groups the white lines always accompany arterio-venous com-

pression, and sometimes they are present alone, especially in the first group. In the diabetic group, in a few cases, I saw the veins depressed without being accompanied by white lines.

I shall first try to describe and explain the formation of the most striking phenomenon—the centripetal deflection of the vein.

In the right eye of a man whose fundi I examined after his death as reported when speaking of the venous pulse, I saw on March 26, 1927, the main branch of the inf. cent. vein crossed near the disc, but still on the retina, by an arteriole or capillary, nearly invisible, a branch of the main artery. The vein was crushed, with white lines at the point of crossing; farther on the vein crossed over the artery, was deflected centripetally and narrowed. The vein was enlarged peripherally from this last point. On May 15, 1927, I noticed that the vein was deflected centripetally by the arteriole, it was now narrowed and was in contact with the main artery at two places. On August 7, 1927, the vein was already quite close to the artery. On November 19, the vein was touching the artery. The arteriole increased in its visibility and was very clear on November 13.

The next case is a man aged 63 years, blood pressure 240-265/130-150, with definite nephro-sclerosis. His right fundus is reproduced in illustration No. 22, and the left one in No. 23, both made on January 13, 1928. White lines are exaggerated for the sake of contrast. They were best seen with red-free light. I shall quote from his history only the facts concerning the centripetal deflection of the veins. The first examination of the fundus was made on December 2, 1926. Seen and examined many times afterwards. On September 1, 1927, I noted that the right sup. temp. vein was crossed by a small arterial branch (I thought this was the first crossing, but later on another crossing appeared before it; it is the second crossing with the centripetal deflection of the vein in the drawing) (vena 10, *a. I*, their approximate volumes). The vein was sharply deflected centripetally, without interference with the blood stream. In the left eye, the artery (sup. temp.) only adjoined the vein, which was depressed downwards, without any interference with the blood stream. On November 17, 1927, I noted, " the right sup. retinal vein is crossed (1) by a tiny artery which can be seen only between the artery and the vein and which is already making a notch in the internal side of the vein, looking away from the artery (first crossing in the illustration—v. 20, *a. I.*); (2) by the artery seen on September 1, 1927; (3) also by a small arterial twig, making a notch in the internal side of the vein. In the left eye, a striking change is seen in the lumen of the a. ret. inf. and its branches—they are covered with a white veil." On November 27, 1927: " R.E. a striking change in the lumen of the sup. temp. artery (also shown in the illustration between the first and third

crossings). Haemorrhages in the retina are seen near the crossings noted on November 7, 1927." On January 13, 1928, the present illustration was made

The case, illustrated in drawing No. 24, is that of a woman aged 53 years, blood pressure 210-220/110-115. On March 20, 1927, I noted "the right sup. temp. vein is crossed four times by the artery; at the first crossing it is crushed, without being deflected; at the second, it is sharply deflected centrifugally; at the third and fourth points a branch of the vein is crossed without any change at all." On September 7, 1927, six months later, I wrote " at the third crossing the vein is deflected centrifugally (this was not seen six months before); at the fourth crossing is commencing a crushing of the vein." On November 3, 1927, " at the first crossing the vein is crushed; at the second the vein is crushed and deflected centrifugally; at the third, the vein is crushed and deflected. A small arterial twig deflects the main vein centripetally. Farther on the vein is divided into two branches; the lower one is deflected centripetally by the artery." I purposely give the descriptions of my note-book, to substantiate myself. I always first examined afresh the fundus and wrote down my observations, and only afterwards did I compare them with my previous notes. The illustration was made on January 13, 1928.

What I mean by centripetal deflection of the vein is demonstrated in the following illustrations:

No. 35. The first branch of the sup. temp. vein at its second crossing by a small arterial twig.

No. 21. The second branch of the sup. nasal vein is deflected and banked by an artery.

No. 20. The sup. nasal artery on the disc is deflected by an artery.

No. 18. The inf. vein is deflected by a tiny artery.

No. 25. The inf. temp. vein crosses the artery once—it is narrowed and centripetally deflected.

No. 10. The sup. nasal vein is deflected by the first artery crossing it on the retina.

No. 26. The sup. nasal vein, a large one, is sharply deflected by a tiny artery.

In Frost's atlas, in Fig. 86, the sup. temp. vein is crossed by the artery and centripetally deflected, without this being mentioned in the text.

Let the reader remember that a centripetal deflection of the vein may also be congenital, simply a wave, and the artery happens to cross just at that point.

Altogether I saw centripetal deflection of the veins in fifteen patients out of over 160 in the first group; in 37 cases out of over 260 in the second; and in the diabetic group in 5 out of 26. It was

usually the artery which crossed over the vein. In only two cases out of 57 did the vein cross over the artery and was centripetally deflected—as in illustration No. 25. Usually in both fundi it was only at one crossing, very seldom at two or more, that this centripetal deflection was seen, the other veins being crushed without a deflection or being centrifugally deflected. In one case I saw the vein crossing over the artery, and even there, in time, the vein became deflected centripetally, a striking demonstration in my opinion, that the perivascularis, which binds the vessels together, and not the artery itself, was the cause of the displacement.

In describing the mechanism of this remarkable phenomenon, I should give an outline of the ophthalmoscopic picture. First one sees a tiny streak of whitish tissue which was not observed previously, passing from the main artery or a large branch to the neighbouring vein. The streak crosses over the vein and makes a notch on the side away from the artery. Gradually one sees this notch press into the venous wall more and more and a centripetal deflection forms slowly. At the same time a tiny, rosy, nearly invisible line, is seen running in the middle of this whitish streak; the streak grows whiter, more distinct and brilliant. At the end of the process, the vein is deflected, the streak marked (white stripes at the point of crossing) and instead of the rosy line a tiny arteriole may now be seen.

We can suppose that the arterial twig is not a newly-formed vessel—it is congenital, and the crossing always existed there. There is no haemorrhage, nor exudation, nor any obstruction to give us the right to suggest the formation of a new vessel. Gravity —the weight of the arterial wall and blood-stream—we can also exclude—it never affects the vein in a normal eye and also does not increase in the sclerotic eye; besides, it is always a very tiny vessel. So we are only left with sclerosis of the arterial wall and sclerosis of the perivascularis, but the sclerotic artery is elongated and not shortened; therefore, it ought to push the vein away from the main artery and not pull the vessels together. Therefore, by *reductio ad absurdum,* we are only left with perivascularis—thrown like a bridge over the main artery and the vein, when accompanying the crossing arterial twig. As a connective tissue formation, when in a state of sclerosis, it will form a scar; it will contract and therefore will pull together the artery and the vein. The vein, the weaker of the two, will be bent and drawn nearer the artery. That is why first is seen the whitish streak of tissue, gradually increasing in its intensity; that is why it first makes a notch in the vein, then squeezes in the wall and deflects the vessel. That is why also the arteriole is not seen at first but is seen later, when the contrast with the white perivascularis is greater.

Sclerosis of the perivascular channel of the arteriole is the cause of the centripetal deflection of the vein. It takes some time to develop.

Centrifugal deflection of the veins, when crossed by the arteries, or even when crossing over them, is of more frequent occurrence. Sometimes, it is accompanied by crushing and banking—signs of actual impediment of the venous circulation—sometimes not. It is difficult to decide which occurs first—the crushing or the deflection. If, for instance, we take all the cases, where the veins are crushed with or without interference, they will form in the first group a total of 26 per cent., in the second 58 per cent., and in the diabetic group 55 per cent. The corresponding figures for all deflections of the veins, centripetal and centrifugal, will be 26 per cent., 50 per cent., and 60 per cent.; that is, nearly the same. The crushing may be of a moderate degree or may be very extreme, what I call an " hour-glass " contracted vein. This, of course, is always a sign of very advanced sclerosis. In following up the same case, one can see that this crushing increases with time. The same refers to the deflection—it certainly increases with time, but not in all cases. It is remarkable that often one particular spot of a vessel is more attacked than the rest. By close study, one comes to the conclusion that most of the manifestations of sclerosis are *local* in origin for a particular fundus, that it is not represented to the same degree throughout the fundus. This is why in the same eye one part of a vessel may be attacked, and the rest not at all. The only exception to this is loss of translucency. It usually occurs in all the vessels and it is very seldom that a few of them still remain translucent. In a few cases I saw a simple crushing of the vein transformed, later on, into a crushing and centrifugal deflection of the vein, but I never saw a centrifugal deflection transform into a centripetal one, and *vice versa*.

In de Schweinitz's book, in plate No. 4 (retinitis albuminurica), the sup. temp. vein is depressed, crushed and banked by the artery. It is the same with the inf. temp. artery. A tiny artery crushes and banks a venous branch of the sup. temp. vein. The veins are not deflected. In Frost's atlas banking and crushing of the veins are seen in drawings 14, 74, 75, 80, and 84. Of the illustrations of this paper, No. 10 shows an " hour-glass " contracted inf. cent. vein without any displacement. The sup. nasal vein is deflected centrifugally, but is less crushed. In No. 25 the inf. nasal vein is " hour-glass " contracted and is commencing to be centripetally deflected. In No. 18 the sup. temp. vein is " hour-glass " contracted, without any displacement. In No. 14 the sup. temp. vein is slightly crushed at the second crossing; the sup. temp. artery is a good demonstration of what I previously called " not a true silver-wire " artery. In No. 17 the sup. temp. vein

is " hour-glass " contracted without any displacement. The same occurs in No. 9.

In my own cases the " hour-glass " contracted and very banked veins are shown in illustration No. 27, without any appreciable deflection; the same is seen in No. 23 (peripheral crossings of the sup. temp. artery). Highly instructive are the different crossings of the same two vessels in No. 20.

The veins cross over the arteries, but are still deflected centrifugally. This is shown in my illustrations Nos. 23, 34, 35.

All these changes at the crossings are pathological, produced by sclerosis. What are the elements in the process?

(*a*) The vein—this is always the weaker partner—the sufferer. It shows by its crushing, deflection or collapse the degree and character of the pressure under which it is. One would think that sometimes a very large vein would be able to crush a tiny artery by the sheer weight of its blood column; we can discard this suggestion at once, because it never happens in the normal eye; but it may happen in the sclerotic one, though very seldom. (See below the chapter on aneurysms). In this case, evidently, the fault lies with the venous wall and its perivascularis.

(*b*) The blood-column in the artery itself. It cannot affect the vein at all, because, similar to the venous blood-stream, it is practically the same in the normal and sclerotic eye.

(*c*) The sclerotic arterial wall. This is the obvious cause. Thickened, hardened, bent as it elongates, the arterial wall of a sclerotic artery always crushes the vein by pressing on it, when *going over* the vein, and deflects it centrifugally in the direction of the arterial blood current and the direction of the elongation of the artery; but this does not explain why it may narrow and deflect centrifugally the vein, when the artery passes under it. It does not explain why the same vessels at different crossings have various changes, as the effect of the sclerotic artery is always the same—crushing and centrifugal deflection of the vein.

(*d*) Here comes into the picture the sclerosis of the perivascularis. For the purpose of illustration I have drawn this imaginary scheme. Suppose the vein runs through the channel A.B.C.D. and the artery through the channel C.D.E.F., or *vice versa*. This connective tissue channel, when in process of sclerosis, will contract in all directions, as does every fibrous tissue. It will contract in the vertical meridian, pulling together the line A.B. with the line C.D. The line C.D. will be the central point; it is the most powerful one, because it is formed by the meeting of two walls of perivascular channels; moreover, the actions of A.B. and E.F. are antagonistic, as each will pull C.D. to its own side. The result will be that the lines A.C.E. and B.D.F. will be shortened; they will press on the vessels inside the channels, they

will squeeze, and narrow them. The artery, with its strong muscularis media, will withstand the pressure from outside, without suffering any injury. The vein, with its collapsible wall, will collapse inside the channel and will bank just outside it. *The natural tendency of the sclerotic artery to crush the vein will be increased by this narrowing of the perivascular channel,* and the vein will be narrowed, even when it crosses over the artery.

The channel will contract also in the horizontal diameter, the lines A.C.E. and B.D.F. will try to meet—the lines A.B., C.D., and E.F. will be shortened. Again the artery will withstand this with impunity, but the vein will be deflected in the direction of this contraction. This contraction will direct itself from the periphery to the centre, towards the disc, as the perivascularis increases in thickness in this direction, or, to be more correct, it decreases with the size of the vessel. Therefore, the natural tendency of the vein is to be deflected *centripetally* by the sclerosis of this channel, and this we have seen, when we discussed the centripetal deflection of the vein, when this channel is unhampered, because the arteriole is too weak to oppose it. In the crossings of large vessels this tendency comes into conflict with the large artery, which is bent as it elongates and deflecting the vein centrifugally. Should the artery be the more powerful or, perhaps, should its wall become first sclerotic—the vein will be crushed and deflected centrifugally. Should the perivascularis have the greater effect—the vein will be crushed and deflected centripetally; should both opposing forces neutralise one another—the vein will be crushed, but not deflected. *The natural tendency of the sclerotic artery to deflect the vein centrifugally will be counteracted be the natural tendency of the perivascular channel to deflect it centripetally.*

Those are the factors in the play. Their interaction will explain, in my opinion, all the modifications of arterio-venous compression. This will also explain why " the vein takes the shortest route (Foster Moore)." It simply follows the contraction of the perivascularis. Sclerosis, then, does not create new factors—it simply operates on a pre-existing condition and aggravates it. Therefore, *a priori,* it ought to be more evident on the disc, where

the vessels are compressed even in a normal eye—and so it is in life—the vessels in a sclerotic eye being more *crushed* on the disc, than on the retina, but not *deflected*.

I have now to say a few words about minor points. Is the venous circulation really impeded? Certainly it is. This is testified by the fact that often the venous pulse is interrupted. This is seen from actual " banking " of the vein, which increases in the same person with the progress of the sclerosis. This is proved in extreme cases by actual collapse of the vein, as is seen in illustration No. 27. True enough, the impediment is not so severe as to produce haemorrhages or oedema of the retina; but still it exists. If the observer will notice the general direction of the vein before and after the crossing he will see that in the majority of cases this is not altered, and therefore centrifugal and centripetal deflection is a purely local condition, caused by local sclerosis, and does not interfere with the general direction of the course of the vein. Sometimes, he will find the peripheral end of the vein deviated to another level than the proximal end. What is the cause of this? Simply, in my opinion, the vein changes its direction *per se* at this point, congenitally; the artery happens to pass over this spot and adds a local deflection through sclerosis, but it cannot, obviously, cause a displacement of the whole peripheral portion of the vein.

Important is the fact that, once formed, those vascular changes never disappear. The blood pressure may become normal, but the perivascular and vascular wall changes will remain as silent witnesses of the vicious past. That is why it is so difficult sometimes to decide, in the aged especially, when one sees an advanced sclerotic fundus and normal blood pressure, whether we are dealing only with involutionary sclerosis; or whether hypertension was present in the past.

Last year I had to attend a case of miscarriage. The bleeding was so severe that at one time the systolic blood pressure fell to 30 mm. The reader will understand that I did not feel very happy, although in the end the woman recovered. When I examined her fundi, with the systolic blood pressure at 30 mm., I saw the arteries very contracted, very rosy, but the crossings, especially on the disc, still present without any appreciable change. It was the same with the fundi that I examined after death. One is inclined to think that pure vascular wall changes would not be so constant, but change of the perivascularis would.

Aneurysms

" Aneurysm of the retinal arteries is so uncommon that it may be looked upon as an ophthalmoscopic curiosity. Both the fusiform and sacculated variety have, however, occasionally been seen."

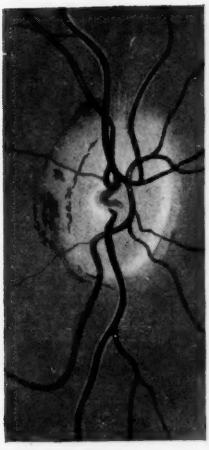


FIG. 7.

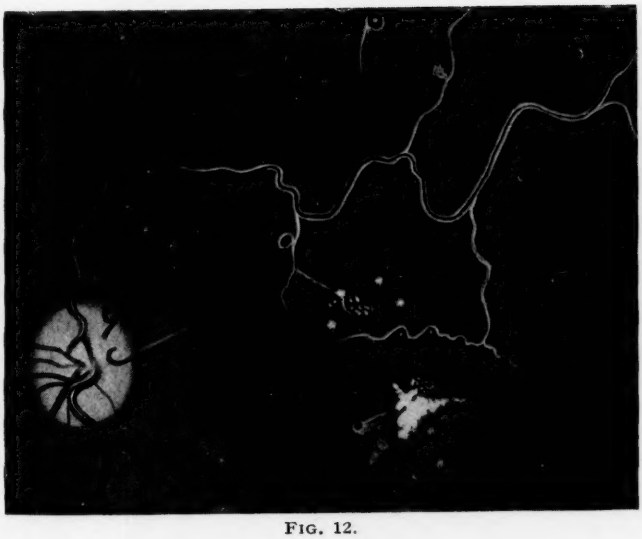


FIG. 12.

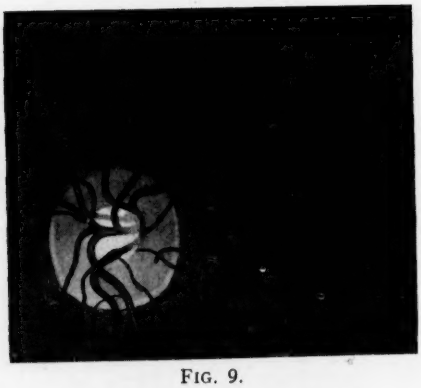


FIG. 9.

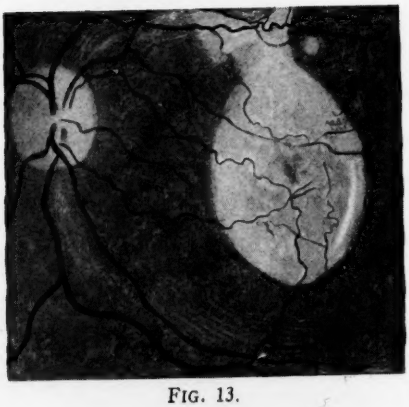


FIG. 13.

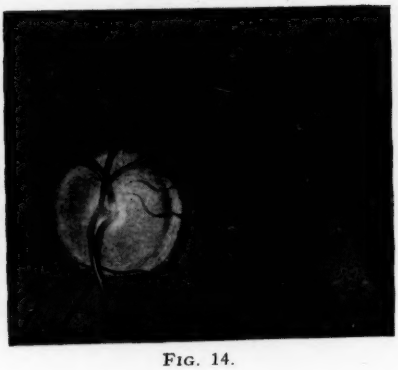


FIG. 14.

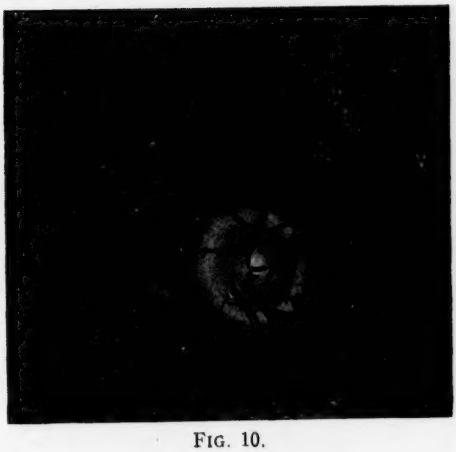


FIG. 10.

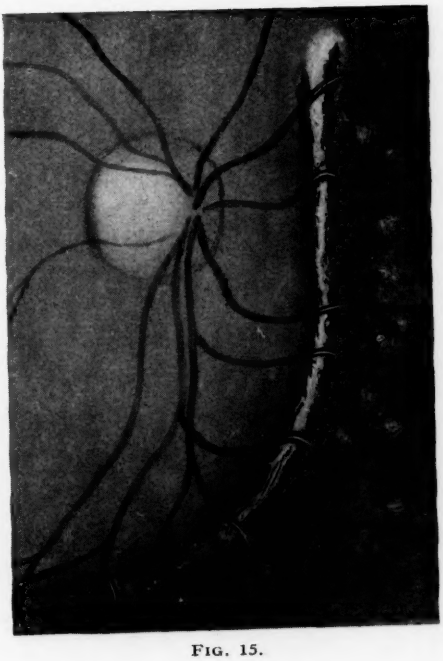


FIG. 15.

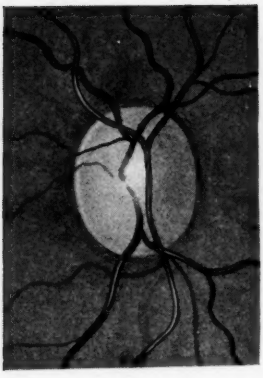


FIG. 11.

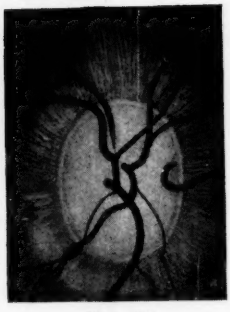


FIG. 17.

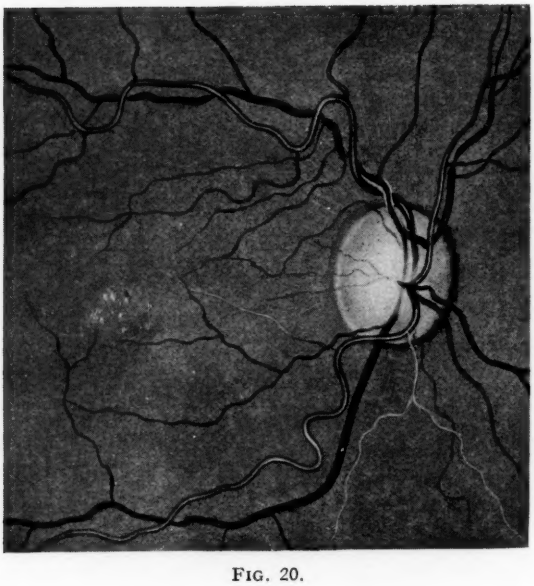


FIG. 20.

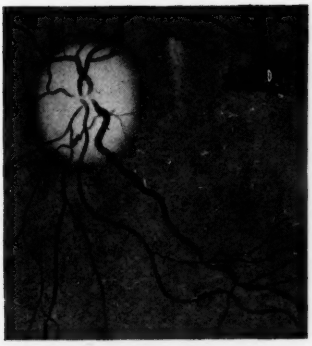


FIG. 18.

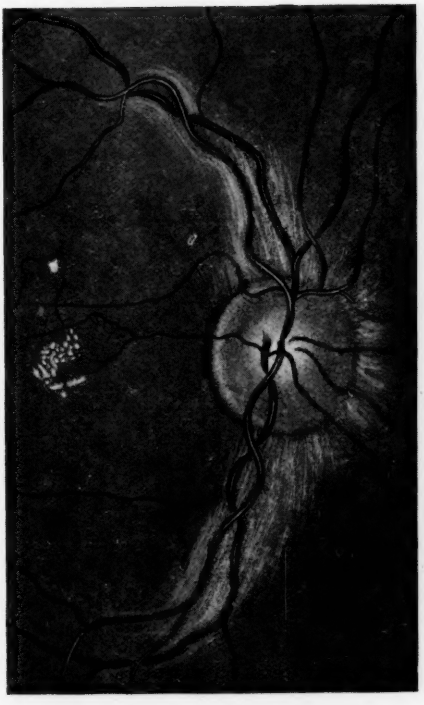


FIG. 19.

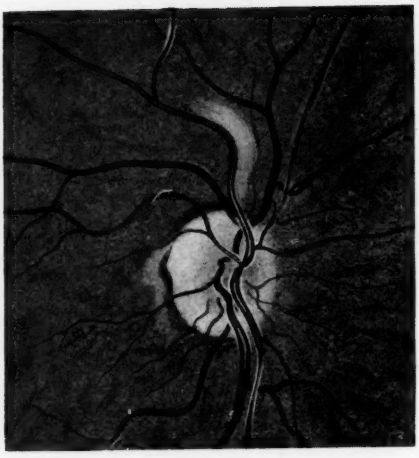


FIG. 21.

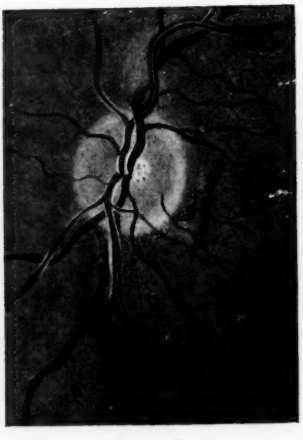
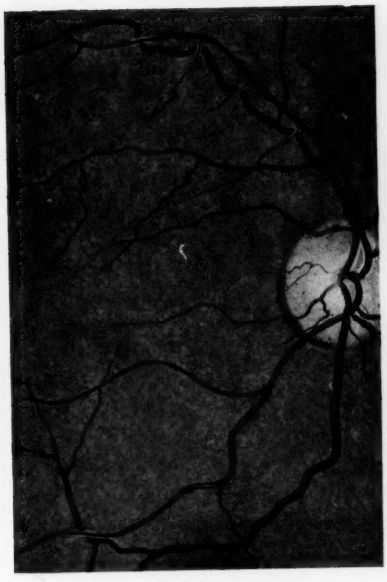


FIG. 27.



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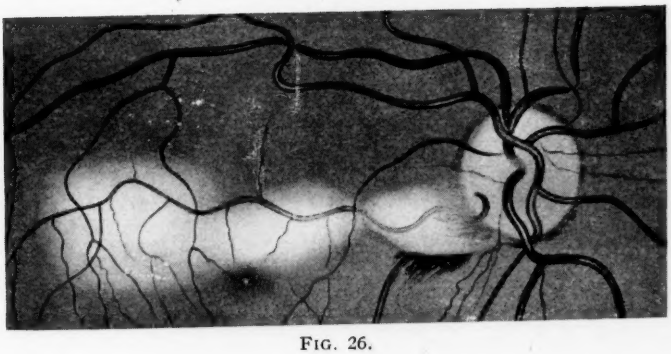
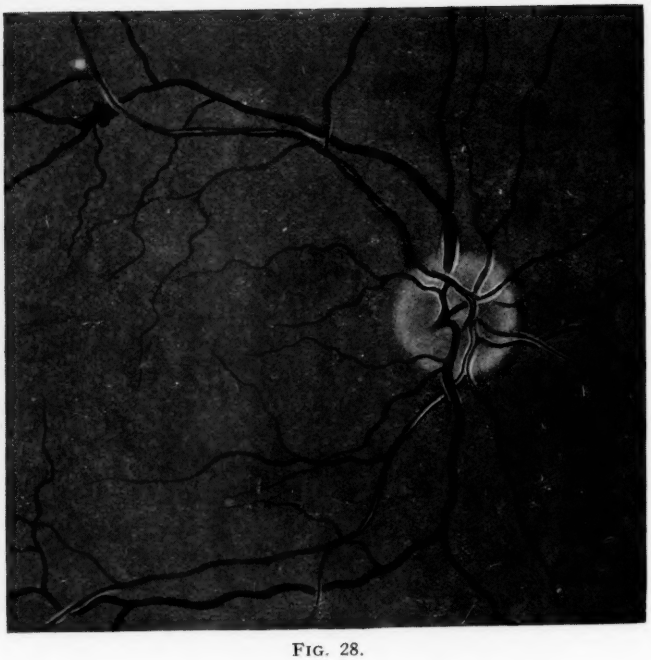


FIG. 26.



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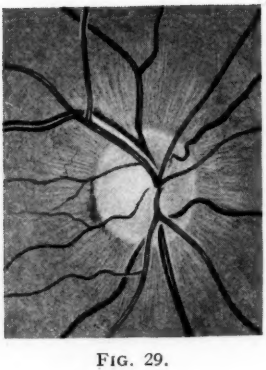

FIG. 29.

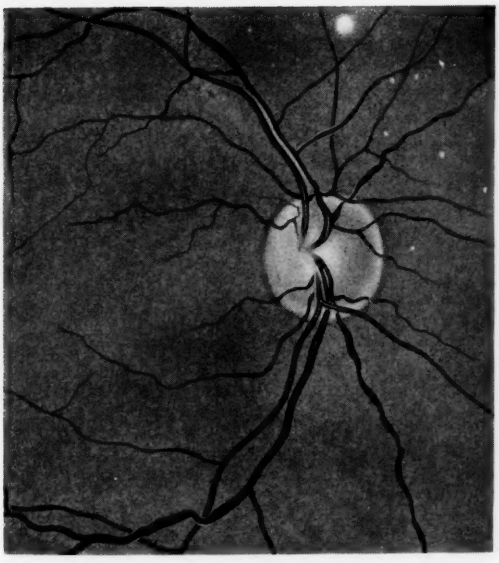

FIG. 35.

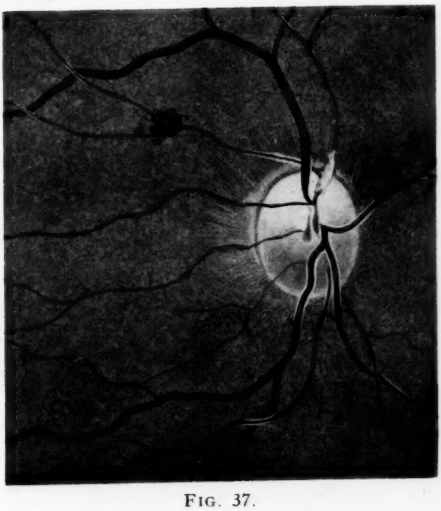

FIG. 37.

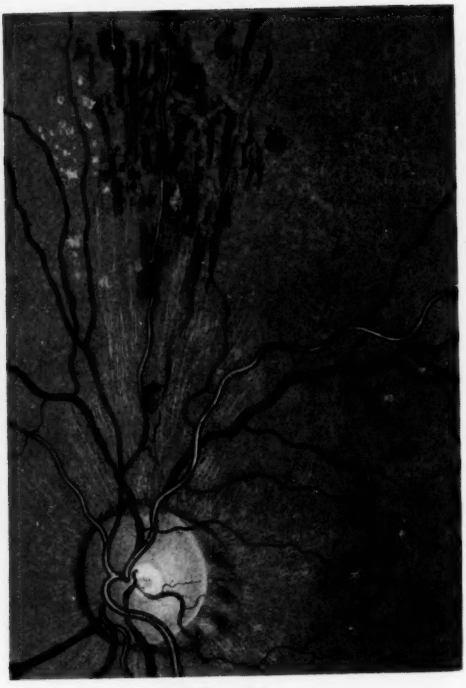

FIG. 36.

(Frost, p. 162.) " In the case of Story and Benson it was probably due to degenerative changes, occurring in the vessel wall, in consequence of retinitis." (*Ibidem.*) " The common form of cerebral haemorrhage is due to the rupture of miliary aneurysms, that is, minute arteries suffer in the nutrition of their walls which yield before the blood pressure and dilatations are produced." (Gowers, p. 135.) " They (aneurysms of large retinal vessels) are probably congenital in origin." (Foster Moore, p. 230.) "The danger of rupture of the aneurysms is not great." (Graefe-Saemisch, p. 24.) " The cause of all aneurysms is always a rupture of the atheromatous wall—a false aneurysm." (*Ibidem,* p. 25.) " The chief cause is fragility of the arterial wall (calcification). The blood pressure is only a secondary factor." " First is ruptured the intima, then the media and the blood is under the adventitia—dissecting (*differzierenden*) aneurysm. If the adventitia also ruptures the blood collects in the perivascular lymphatic space—spurious aneurysm. Pick and Ellis show that cerebral haemorrhage is caused either by the rupture of aneurysms other than larger miliary ones, or directly by the rupture of arteries.". . . (*Ibidem.*)

In my cases, I saw miliary aneurysms only; one on the disc and one on the retina in the first group, one on the disc and three on the retina in the second. I am inclined to think that they are very rare on the disc, and I am under the impression that Gunn, in Gowers' book, is of the same opinion. It is not always easy to decide whether what one sees is an aneurysm or a haemorrhage. Once, on the disc, I saw the following picture; a tiny vein, with a knot in the middle, joining the main vein. The patient was a sclerotic, with a blood pressure 210/100, with two paralytic strokes in his past. He was a man aged 60 years. One would think that this knot was simply a point where two veins met, one of them coming up from the tissue of the disc, but then the proximal end, between the knot and the main vein, ought to have been larger than the distal one, and it was not. So I was left with the conclusion that this was a true venous aneurysm, caused most probably, by a local atheromatous focus in the venous wall. In illustration No. 34 is seen a small venous aneurysm, like an opened umbrella over a venule. It has been there without any change for two years. Is it not possible for it to have been formed, like a true aneurysm, by dilatation of the venule, if the blood-stream is impeded? In illustration No. 31, the sup. temp. artery crosses and crushes the vein. The vein shows two aneurysms or perhaps haemorrhages, as the contours are not distinct; but they are certainly aneurysms in illustration No. 32. The explanation is obvious. The nutrition of the venous wall is so much impaired by the crushing that it gives way under the pressure of the blood-

stream and there forms, most probably, a true aneurysm, before the crossing.

What would happen in a similar case to an artery? Imagine the sclerotic wall and chiefly the perivascularis of a very large vein crushing a small and narrow artery. The muscularis media will resist until the last and will react to the injury by contraction. Therefore, instead of being enlarged before the crossing, as the vein is, the artery will be contracted, but the crossing will impair the nutrition of the wall of the artery. Therefore, the arterial wall after the crossing will be weaker. The blood stream emerging from under the vein will exert its pressure in all directions. The weakened part of the arterial wall will not be able to withstand this pressure and will dilate. Most probably, a true fusiform aneurysm will be formed. This kind of venous and arterial aneurysm which is not described anywhere, as far as my knowledge goes, I call "aneurysm by crushing." An essential condition for its creation is sclerosis of the perivascularis, contracting and pulling down the vein on the small underlying artery.

But will the blood current really emerge from under the crossing with some force? On p. 85 in Bailliart's book will be found the answer: "If you will examine the conditions of a liquid running out of a tube with a tap you will see that when the tap is more open the pressure of the liquid and the speed of its running increase after passing the tap, and its pressure is less and the speed greater before it; and when the tap is less open than previously, the pressure of the liquid and its speed are less after the tap, and the pressure is greater and the speed less before the tap." Theoretically the arterial pulse ought to be present more frequently in retinal aneurysm, but as a matter of fact, it is not. Leber (Graefe-Saemisch, p. 23) also points out, as a noteworthy fact, that the spontaneous pulse is often absent in retinal aneurysms.

What I believe to be the earliest stage of such an aneurysm I saw in a man aged 36 years, with a blood pressure of 110/60 and aortic regurgitation. Translucency of his retinal arteries was lost, copper-wire colour, light reflex dotted. In the right eye, the first branch of the sup. temp. artery was crossed by a vein (*a. I, v. 3*), the artery was crushed, with a change in its lumen, and was slightly pulsating. The reader will realise that to see pulsation in a very small artery when, on the disc, the arteries were not pulsating, was a striking phenomenon. Still, it existed, and afforded me the best proof that crushing of one vessel by another means an actual impediment of the vascular circulation.

The other case was that of a lady aged 51 years, with a blood pressure 165/100. The translucency of her retinal vessels was lost; copper-wire colour, light reflex dotted; white lines at the crossings. The veins were crushed by the arteries with inter-

ference of the blood-stream. In the right eye the sup. temp. vein was crushed three times by the artery and showed an ampulliform dilatation between the second and third crossings. A small branch of the sup. temp. artery was crossed by the vein not far from the disc. The artery was crushed and had an ampulliform dilatation peripheral to the crossing, where it was very thin. The arterial pulse was visible before the crossing, other arteries did not show it. I saw her again eight months later. The arterial pulse was less pronounced, practically gone. Aneurysm present.

The third example was case No. 30. Seen first on May 25, 1926, aged 55 years, blood pressure 210-250/140-130. A paralytic stroke in the past. Translucency of the vessels lost, copper-wire, in some places silvery-wire colour; light reflex dotted. The veins were crushed by the arteries with interference of the blood-current and deflected centrifugally. In the right eye the sup. temp. vein was crushed by the artery. Farther on one arterial branch crossed *under* the vein; the vein was decidedly elevated and sharply bent centrifugally. A small branch of the inf. cent. artery in the left eye (seen in the illustration) was crossed by a large inf. cent. vein. The artery was crushed with an aneurysm on the peripheral side of the crossing. No arterial pulse was seen. The illustration was made on January 13, 1928, and all this time the aneurysm remained stationary. It is quite probable that in the brain, where the neuroglia and the perivascularis are much stronger than in the retina, such aneurysms are produced more often than in the retina.

Conclusions.—As far as my cases show, a true aneurysm may be seen on the retinal vessels; it is generally of the miliary type. In the case of veins it is formed by dilatation of a venule with impediment of the blood-stream—in arteries by interference with their nutrition; the danger of rupture is not great.

Retinal Haemorrhages

One old retinal haemorrhage was seen in group No. 1, where no fresh haemorrhages were observed at all. In group No. 2, I saw fresh retinal haemorrhages in 25 patients (9 per cent.) and old ones in 7 patients (2.5 per cent.). In the diabetic group, there were fresh haemorrhages in 4 patients (15 per cent.) and old ones in 2 patients (7 per cent.). I did not include here two cases of venous thrombosis. This would bring the number up to 27 cases in group No. 2. The fact of such numbers is very suggestive—enter arterial hypertension and the number of retinal haemorrhages increased considerably—a noteworthy fact that has been mentioned many times previously.

The first and most probably the deciding factor is local degener-

ation (atheroma) of the retinal vessel. This must be very marked indeed, as the walls are supported by the perivascularis and by the intraocular pressure. This may be the explanation why retinal haemorrhages are so seldom seen in children with whooping cough, although severe subconjunctival haemorrhages are a common occurrence. Gowers, p. 28, says that " degeneration of the retinal vessels is probably the most potent cause of haemorrhage, although it cannot always be demonstrated post-mortem." This is also the opinion of Leber (Graefe-Saemisch, p. 498). As in the case of aneurysms, fragility of the vascular wall increases, according to my table, with increase of the arterial tension—*i.e., atheroma is more often seen in combination with essential hyperpiesis than with ordinary involutionary sclerosis.* Again essential hyperpiesis plays here the rôle of a primary factor, and vascular changes proper are only secondary to it. The connection between retinal haemorrhages and arterial hypertension was known long ago, but the increased blood pressure was always regarded as a secondary factor. " The chief cause is fragility of the arterial wall (calcification); the blood pressure is only a secondary factor," says Prof. Leber, p. 26, when describing the origin of miliary retinal aneurysms. " General increased blood pressure seems to be an occasional cause, for high arterial tension may exist when no other cause can be discovered. . . . But it (the retinal haemorrhage) is, on the whole, a rare accident, considering the frequency with which high tension exists." (*Ibidem.*) As far as my cases show, essential hyperpiesis probably first prepares the way for a haemorrhage by local degeneration of the vascular wall and then actually provokes the bleeding.

How does this bleeding occur? Very seldom, if at all, by rhexis, as the vessels are surrounded by strong perivascularis; more probably by diapedesis. However, in one case of fatal albuminuric retinitis, I saw a large pre-retinal haemorrhage, which I think, proceeded from the inf. cent. vein where crossed and crushed by the artery. In two cases, I think, I saw the actual process of diapedesis. In one case (in group No. 2) I saw a small artery, when crossing a vein, accompanied by two rosy lines just outside the vessel; the artery showed double contours of its wall, imperceptibly passing into the retinal tissue. Gradually, in a few days' time, the infiltration increased and the rosy lines were transformed into the usual deeply coloured lines of haemorrhage. In the other case (also in group No. 2) with a haemorrhage on the retina below the disc, I saw a peculiar veil of dirty colour, covering a point where the sup. temp. artery divided into a few branches and at the same time crossed the sup. temp. vein. It differed by its colour from ordinary exudation; it never developed into a proper haemorrhage, and disappeared in a week or two,

leaving behind a slight change in the lumen of the vein. I think it was a slight sanguineous exudation.

Usually the haemorrhages proceed from the veins, but in a few cases I was under the impression that they were arterial in origin and twice I could prove it. In one instance, a case of retinitis albuminurica, I saw on the inf. cent. artery which was oedematous and swollen, 2-3 disc diameters away from the papilla, just outside the blood stream, but still segregated from it by a whitish line, between it and the perivascularis, a red ring appear around the vessel. From the perivascularis it was also segregated by a whitish line. It was very thin, bright red and very short. It completely disappeared in a week or so. I think that it was an extravasation of blood between the adventitia and perivascularis.

The other case is represented by illustration No. 36. A branch of the sup. nasal artery divides into three small, narrow, but very long arteries. The nasal one runs upwards, dividing itself again into two branches with foci of retinal exudation in its area. The middle branch is dark, thrombosed. (This was better seen in the patient than in the illustration.) The third one is very faintly seen on the temporal side. The nearest veins are the nasal vein, dividing into two, and a small venous branch of the sup. temp. vein. As the middle artery was thrombosed and its area was covered with haemorrhages I think that I can assert their arterial origin, the veins being far away.

It is rather curious to see how sometimes pathology points out some unknown or undecided anatomical facts. We saw this when describing the disc of a normal fundus. Prof. Leber does not mention the work of Krückmann and others; for him the retina has only the membrana limitans interna, but on p. 507, when describing some peculiar forms of retinal haemorrhage with separation of the membrana limitans interna, he wonders what is the intimate structure of this membrane, and thinks that it is composed of two layers—the membrane proper and under it—Müller's radial fibres and their endings. On p. 508, Leber admits that there is an intimate connection (*innige verbindung*) between the membrana limitans retinae and the glia at the border of the disc.

In Fuchs' text-book, Edit. 1911, I found a statement that retinal haemorrhages were so common with malignant disease of the stomach as to help in the diagnosis; in a few cases of definite carcinoma of the stomach I did not see them.

Speaking on the classification of retinal haemorrhages at the conference of the Brit. Med. Assoc. in 1926, in an excellent paper, Foster Moore warned the profession that a sudden considerable fall in the blood pressure is not safe from the ophthalmological point of view. It may cause retinal haemorrhages. This is correct for

a local reduction, for instance, when operating in glaucoma, but I wonder if a lowering of the general blood pressure would affect such a jealously autonomous organ as the eyeball. Many times I saw the blood pressure fall 100 mm. in 24 hours, often by a liberal venesection, but never observed this to cause a haemorrhage. On purely theoretical grounds one would rather expect a thrombosis in the eye, as occasionally happens in other parts of the body.

In the same paper the reader can see for himself how important it is to divide involutionary arterio-sclerosis and essential hyperpiesis as clinical entities. Foster Moore says (*Brit. Med. Jl.*, December 11, 1926), " It is, of course, well recognised that folks with a high systolic pressure, for example, in renal disease or primary arterio-sclerosis, frequently have retinal haemorrhages." No mention is made of the diastolic pressure at all. But what really is primary arterio-sclerosis? If it be essential hyperpiesis, then this disease has nothing to do with arterio-sclerosis. If Foster Moore means involutionary arterio-sclerosis, then this disease is not accompanied by raised arterial tension. I am inclined to think that he means " essential hyperpiesis " and he should not use the misleading term of " arterio-sclerosis." The same happens with this writer on p. 85 of his "Medical Ophthalmology." He rightly ascribes thrombosis of the veins to arterio-sclerosis (atheroma, I should say) by pressure of the sclerotic artery. But farther on he evidently does not differentiate between essential hyperpiesis and arterio-sclerosis. " In addition the systolic blood pressure in 40 of the 61 patients, on whom it was taken at intervals, was on an average 180 mm., in 11 only was it less than 160." It is the same with Maitland Ramsay. On p. 397 he says: " Arterio-sclerosis may show itself very early in the retinal blood vessels and give rise to haemorrhage long before there is any manifest renal inadequacy." A perfectly correct statement, but instead of " arterio-sclerosis," " essential hyperpiesis " ought to be printed.

What is the prognosis of retinal haemorrhages? Gunn, at the meeting of the Ophthalmological Society of the United Kingdom, after reading his famous paper answered a question of MacKinlay by saying that he thought that retinal haemorrhages are chiefly the probable signs of an approaching cerebral haemorrhage. This is a very pessimistic view. Foster Moore on p. 55 in his book says, "The retinal haemorrhages are incidental to the arterial disease and their presence in general does not convey any specific significance; in some cases, however, an increase in their number precedes the onset of serious symptoms." This is a very optimistic view. The truth, in my opinion, lies in between. Perhaps nowhere else in medicine can we see a better illustration of the rule that " all

depends on the circumstances of place and time." If fresh retinal haemorrhages appear in spite of a lowering of the blood pressure in essential hyperpiesis, the danger is great; if they appear in diabetes and if in spite of insulin the blood pressure is rising and the specific gravity of the urine is decreasing then the danger is great.

But, we can occasionally hear it said " A retinal haemorrhage is so small! And often nothing happens to the patient for years! " If we compare the amount of blood running through the eye-ball with that running through the lung, the smallest retinal haemorrhage will amount approximately to half a teaspoonful of blood in the sputum. And surely such a haemoptysis will move a physician to undertake a thorough investigation of the pulmonary system, in spite of the fact that often nothing happens to his patient for years after he has coughed up blood. The retinal haemorrhage is always a serious and temporary warning, although it does not always mean immediate danger. And I earnestly wish that its significance should be grasped and should lead to the same thorough investigation of the reno-cardio-vascular system, as a haemoptysis usually does for the lungs.

Summary.—As far as my investigations have shown, in spite of the lack of direct anatomical evidence, the vessels in the normal eye run on the disc and on the retina through channels of perivascularis. The disc is covered by a transparent and strong membrane of mesodermic origin. The vessels on the disc are under pressure in the sagittal and horizontal diameters, which is greater than that on the retina. When they become sclerotic they first lose their translucency, then change their colour, and " dot " their light reflex, then the sclerotic perivascularis appears, first as white lines at the crossing points, and later on it takes a leading part in arterio-venous compression. This thickened perivascularis plays an important part in every pathological vascular process in the sclerotic eye, but its action is chiefly a local one affecting separate parts of the vessel, especially at crossing points. Essential hyperpiesis greatly increases and aggravates all the sclerotic signs of the retinal vessels. It is quite probable that there is a special predilection for the retinal vessels and that these are affected more early and more severely than other parts of the vascular tree, except the cerebral vessels and, perhaps, those of the kidneys.

(To be continued.)

CORRIGENDUM

In Dr. Pines's article in our last number, on p. 97, in the penultimate line, for "sum" read sun. On p. 112, line 30, for "Figs. 1, 1*a*, 1*b*," read Figs. 1, 1*a*, 1*b*, 1*c*. On p. 115, line 14, for " endothelial cells (C)," read endothelial cells (B).

ANNOTATION

Ophthalmic Benefit

We quote the following statement of the arrangement arrived at by the British Medical Association and the Association of Dispensing Opticians from the Supplement to the *British Medical Journal* of February 2, 1929.

OPHTHALMIC TREATMENT ON THE CLINIC SYSTEM.

" This Board has decided to establish in large centres a scheme for the provision of ophthalmic benefit (*a*) at central clinics, and (*b*) at home clinics at the ophthalmic practitioners' consulting rooms at fixed hours. This arrangement will provide for ophthalmic examination and advice—that is, examination and prescription for glasses, if required, such operative treatment and advice as could be given at a single consultation at the consulting rooms, and a report, when necessary, for the guidance of the private practitioner, for a fee of 10s. 6d. per case. Any approved society recommending an insured person, entitled to ophthalmic benefit, to receive this benefit under this arrangement, will be required to provide the fee of 10s. 6d. payable to the ophthalmic practitioner, and a further 7s. 6d. in respect of the glasses, where these are prescribed—making a minimum liability of 10s. 6d. and a maximum liability of 18s.; where glasses are not prescribed the total liability is 10s. 6d. for the ophthalmic practitioner's fee.

For the appropriate fee the patient will be supplied without charge with one of the following: Best quality white metal non-rusting rimmed spectacles, best quality white metal non-rusting rimmed eyeglasses, or best quality white metal non-rusting rimless eyeglasses—glazed with any lenses ordered on prescription (with the exception of Crookes's glass, tinted glass, prisms or bifocals, the extra cost of which is shown on a schedule), complete with case. For additional and voluntary payments according to a schedule the member may be supplied with rimmed or rimless spectacles or eyeglasses of superior workmanship.

In the first instance it has been arranged that those presenting themselves for examination will be dealt with by the ophthalmic practitioners at their own private consulting rooms at stated hours. Central clinics will only be established in densely populated areas if occasion demands, and in all cases the alternative of treatment at a home clinic in the ophthalmic practitioner's private consulting rooms at stated hours will be given."

For further details, and also for the reasons that have led the Association to adopt the above scheme our readers should consult the original article. Time alone can show how far the scheme will act satisfactorily. If insured persons, as seems likely, avail themselves of the scheme there may be some risk of depletion of the Out-Patient Departments of both special and general hospitals.

ABSTRACTS

I.—CONJUNCTIVA

(1) **Mariotti, Cesare (Bologna). — A very rare and severe form of pseudo-membranous conjunctivitis of streptococcal origin cured by autovaccines. (Sopra una rarissima e grave forma di congiuntivite pseudomembranosa da streptococco guarita con l'autovaccino.)** *Boll. d'Ocul.*, Vol. VII, No. 11, p. 1118, 1928.

(1) A very severe and intractable case of pseudo-membranous conjunctivitis in an infant is described in this paper, and leads up to a discussion of the various organisms which may give rise to membranous or pseudo-membranous conjunctivitis. Further, **Mariotti** discusses the resistance to treatment of cases of conjunctivitis which are secondary to infection of the lacrimal passages. There is a very full review of the literature bearing on similar cases.

LESLIE PATON.

(2) **Morax, V.—A special affection of the conjunctiva, with pseudomembranous exudates, corneal lesions, and recurrent granulomata. (Note sur une affection particulière de la conjonctive caractérisée par des exsudats pseudo-membraneux, des lésions de la cornée et la formation de granulomes récidivants.)** *Ann. d'Ocul.*, Vol. CLXV, p. 401, 1928.

(2) **Morax** reports at length two cases of pseudo-membranous conjunctivitis with unusual associated features. He also quotes in detail a similar case of Lijo Pavia, and refers to one somewhat analogous described by Kalt and Autier. The most striking features of Morax's cases are quoted.

Case I. A female child, aged three years, during her recovery from an angina and bronchitis with fever rising to 40° C.,

suffered from conjunctivitis of the right eye of many weeks' duration with membrane-formation mostly on the upper tarsal conjunctiva; on some occasions the membrane was found to have disappeared, sometimes with the simultaneous development of a more or less pedunculated granuloma (demonstrated histologically); ablation of the granuloma was followed by further membrane-formation or by recurrence of the granuloma. Histological examination of a granuloma excised from the upper tarsus showed at the base vascular granulation tissue containing masses of hyaline substance. There was dense infiltration of the superficial layers of the granulation tissue with cells, mainly lymphocytes, and superficial to this cellular zone was a false membrane composed of a fibrinous layer with scanty cell infiltration.

Case II. A child at the age of four years developed an acute conjunctivitis with false membrane on the conjunctiva, and angina, also with the presence of a false membrane in the throat. Roux's serum was given by injection. Within a few days corneal ulceration developed and rapid perforation of the cornea occurred. For two years the socket continued to discharge. Morax then examined the case and found the shrunken globe hidden by a granuloma. This and the conjunctival sac were covered by a grey false membrane. There was no palpebral oedema, and no adenitis. The other eye was healthy. After about three months' stay in the country the child's general condition was excellent, and there was no false membrane, but a pedunculated granuloma the size of a small nut occupied the orbit. The granuloma was excised without complication. Four months later the socket was free from granuloma and false membrane.

Details of similar cases are quoted from descriptions by Lijo Pavia (Argentine), and Kalt and Autier (*Bulletin de la Soc. d'Ophtal. de Paris,* 1927). The features of these cases are as follows:

1. The development of a *pseudo-membranous conjunctivitis* with acute or sub-acute onset in association with angina, bronchitis and fever or with angina accompanied by the formation of false membranes.

2. The development of false membranes not influenced in the least degree by injections of antidiphtheritic serum.

3. Both eyes affected in two cases, but the disease had a predominating effect in one eye.

4. After a preliminary period of pseudo-membranous exudate of several months' duration, recurrences of the exudate took place.

5. The *cornea was affected* at the onset in two cases, at later dates in the others. Enucleation was necessary early in one case, and in the others the eyeball became shrunken.

6. The appearance of one or more *granulomata* covered with pseudo-membranous exudate, and the exacerbation of similar exudate after removal of a granuloma. Recurrence of granulomata.

Aetiology.—It is an affection of childhood, the oldest case being ten years of age. Three were female, one male. No definite result was obtained from bacteriological examination, excepting the complete absence of evidence of diphtherial infection.

HUMPHREY NEAME.

(3) **v. Rötth, A. (Pécs).—On lesion of the conjunctiva in erythema nodosum. (Ueber Erkrankung der Bindehaut bei Erythema Nodosum.)** *Zeitschr. f. Augenheilk.*, Vol. LXVI, p. 332, 1928.

(3) Apart from iritis and recurrent iridocyclitis with hypopyon formation, it would appear that lesions of the conjunctiva also may occur in erythema nodosum. As complications of erythema exudativum multiforme, conjunctival lesions are by no means rare; indeed three varieties of conjunctivitis have been described in this connection: catarrhal, membranous, and nodular. In erythema nodosum, however, the text-books do not recognise any conjunctival complication, and the literature on the subject contains but few reported cases. In a case under the care of **v. Rötth** conjunctivitis of the nodular variety developed; this is an agreement with the type reported by other observers and differs in no way from the nodular conjunctivitis seen in erythema multiforme. The lesion is thus described: "The part of the conjunctiva lying between the lids becomes oedematous, then a small nodule is formed, surrounded by an injected area. The lesion is confined mainly to the nasal side of the conjunctiva. The nodule extends down into the subconjunctival layers, perhaps also involving the episclera; usually it disappears together with the erythema nodosum within a few days. The prognosis is good; there is no pain and no treatment is necessary."

The fact that the conjunctival lesion of erythema nodosum corresponds to the most common variety seen in erythema multiforme, is of some interest, as some dermatologists consider both diseases to be of the same origin. Interesting too is the question as to the nature of the nodules: erythema nodosum is suspected to be of a tuberculous nature, but the conjunctival nodules cannot be regarded as tuberculous episcleritis. The appearance and clinical course of these nodules make it impossible to regard them as a deep-seated lesion of a stubborn variety such as tuberculous episcleritis.

A. SOURASKY.

(4) **Urbanek, J. (Vienna).—Dermatitis and conjunctivitis due to light. (Lichtdermatitis und eine Lichterkrankung der Konjunctiva.)** *Zeitschr. f. Augenheilk.*, Bd. LXVI, S. 66, 1927.

(4) **Urbanek** draws attention to a periodic dermatitis characterised by a vesicular eruption occurring in exposed parts of the body and associated with considerable itching which appears to be due to the action of light. It is seasonal in its occurrence coming on in the spring and subsiding in the autumn. It is accompanied by a similar eruption on the conjunctiva and may have some association with vernal catarrh. The suggestion is made that there may be some possible relation between light sensitivity and tuberculosis. Möller, for example, classes lupus erythematoides discoides as belonging to the light dermatoses, and Volk and Grosz have noticed a similar skin reaction after intracutaneous injections of dead tubercle bacilli. Urbanek thinks that this may help to explain the occurrence of periodicity in tuberculous eye diseases and the benefit accruing to such cases from treatment by artificial sunlight.

W. S. DUKE-ELDER.

(5) **Lagrange, Henri.—Notes on vernal catarrh and its connection with the neuro-arthritic syndrome of childhood. (Notes sur la conjonctivite printanière et ses rapports avec le syndrome de neuro-arthritisme infantile.)** *Ann. d'Ocul.*, Vol. CLXV, p. 349, 1928.

(5) **Lagrange** states that he considers cases of vernal catarrh ought to be grouped in the class of cases dependent on what Czerny entitles the exudative diathesis. The condition, he states, is one which occurs about the period of puberty, which disappears when puberty is passed, and in which recovery has been helped by the use of glandular therapy. The idea came to him after observing the case of a woman who had a recurring seasonal conjunctivitis after the removal of the ovaries. The administration of ovarian extract was followed by the arrest of the conjunctivitis. In the four cases reported the ages were from nine to fourteen years; lacrymation, photophobia and irritation of the eyes had recurred in the summer months for three or four seasons, with conjunctival injection. Eosinophiles were found in the conjunctival secretion of three cases. It was noted that the testicles were ectopic or small in three. In one there was a family history of maternal syphilis and of hay fever, in another pneumonia had occurred at the age of eight years, and in a third there had been recurrent fever of unknown cause at the age of three, and recently violent pertussis. [These do not appear to have been cases of typical spring catarrh, for although there was a seasonal element,

and in three of them eosinophiles were found in the conjunctival secretion, there were no typical nodules, either palpebral or ocular, except in one which showed some projections at the limbus. Lacrymation, photophobia and sense of irritation were present in three and conjunctival injection in all four. All four showed sensitiveness to pollen, three by the cutaneous test and one by susceptibility to flowers. They therefore appear to belong to the group of hay fever. As the administration of orchitic extract was usually carried out over a period of several months, it would seem uncertain whether the improvement recorded was *post* or *propter hoc*.]

HUMPHREY NEAME.

(6) **Fahim, Girgis Ibrahim (Suez.)—Spring catarrh.** *Bull. Ophthal. Soc. of Egypt*, 1927.

(6) In a trachomatous country such as Egypt it is possible, unless great care is taken, to miss cases of trachoma which are complicated by spring catarrh. Meyerhof drew attention to the importance of this in 1912 at the same Society, though this has escaped **Fahim**, the author of the paper under review.

As is well known the presence of eosinophilia in the conjunctival secretion provides a means of diagnosing the presence of spring catarrh, masked by trachoma. This was first pointed out by Herbert (*British Medical Journal*, 1907). It occurred to the author of the paper under review that there might be an error in diagnosis if the presence of eosinophilia was relied on, as so many Egyptians have either ankylostomiasis or bilharziasis, and in such conditions of helminthiasis there is eosinophilia of the blood, varying from 6 per cent. to 32 per cent. He therefore examined the conjunctival secretion of sufferers from these parasitic diseases and found eosinophilia absent. He therefore is satisfied that the presence of eosinophilia in the conjunctival secretion is pathognomonic of spring catarrh, even in Egypt.

In the discussion Sobhy stated that MacCallan and he had previously investigated this subject, publishing the results in the Eighth Annual Report of the Ophthalmic Section, Department of Public Health, Egypt, 1920, and had arrived at the same conclusion.

A. F. MACCALLAN.

II.—MISCELLANEOUS

(1) **Veil, Prosper (Paris).—Sclero-keratitis successfully treated by the anti-tuberculous vaccine of Vaudremer. (Sclérokeratite traitée avec succès par le vaccin anti-tuberculeux de Vaudremer.)** *La Clin. Ophtal.*, February, 1928.

(1) Veil's case was shown at the February, 1928, meeting of the Paris Ophthalmological Society. The patient was a young woman, aged 21 years, with a family history of tubercle, but without, as far as could be ascertained by careful examination, any personal lesion. Nevertheless, there were scars in the cervical region. The right eye had been, for three years, the seat of relapsing sclero-keratitis; the left eye presented a leucoma adherens. Taking everything into consideration the author concluded that the basis of the case was tubercle and decided to employ the vaccine of Vaudremer. One need not quote the details of the injections, which were painless and did not produce general, local, or focal reaction.

The photophobia disappeared after the third injection, the eye was white and painless after the fourth. At the end of the course of 12 injections the cornea had partly recovered its transparency, though the corneal microscope showed a rich vascularisation of that membrane. The sclera had a dusky appearance. During the treatment the patient put on four kilogrammes in weight, while her general health was much improved. Another course of the treatment is contemplated. Veil considers that this vaccine has a specific action and that its employment is justified in cases with a family history of tubercle. It is interesting to note that he has tried the vaccine in other cases which were completely free from tubercle, in two cases of iridocyclitis of indeterminate origin, and in one case of vitreous haemorrhage in an adolescent. In none of these cases was the vaccine of any service.

NOTE.—A previous case was reported by Veil and Favory to the April, 1927, meeting of the Paris Ophthalmological Society, and was published along with details of the preparation and method of use of Vaudremer's vaccine, in *La Clin. Ophtal.* for July, 1927. It concerned a woman, aged 23 years, who, four years previously, had suffered from what had been diagnosed as tuberculous pleuro-peritonitis, whose father had had pleurisy, and who (*i.e.*, the woman) presented the signs of pure cyclitis with abundant descemetitis. She was treated with this vaccine, but, while the eye condition manifestly improved, towards the end of the treatment

physical signs appeared at the apices of the lungs and the treatment was stopped.

ERNEST THOMSON.

(2) **Engelking (Freiburg, i. B.)—The genesis of xanthomatosis bulbi. (Ueber die formale und kausale Genese der Xanthomatosis Bulbi.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXIX, S. 721, 1928.

(2) **Engelking** has described eleven cases of xanthomatosis bulbi, and performed a series of experiments on rabbits with a view to determine the factors in its aetiology. From clinical, histological, and experimental work he concludes that the condition is due to a disturbed metabolism in the iris and ciliary body, associated with an infection, or a toxin, or with increased intraocular pressure. Two things are essential: first, an increase of cholesterol in the blood, and, second, a local lesion in the eye.

A typical clinical case is as follows: The patient in 1915, being 56 years of age, had a chronic irido-cyclitis of the right eye, with keratic precipitates and posterior synechiae. Two years later the lens became opaque, and the pupil became occluded with exudative material, the eye meanwhile showing no irritation or pain. In 1926 the eye was amaurotic, the tension was very high, the anterior chamber was shallow. Keratic precipitates were evident in quantity. The iris was atrophic with ring synechiae, and a complicated cataract was fully developed. The other eye showed a chronic uveitis with keratic precipitates accompanied by a commencing cataract and vitreous opacities. In 1927 the right eye, which had been ten years blind, showed xanthomatosis of the iris as a circular yellow ring. The iris was very much atrophied, the cataract was hyper-mature, the intraocular pressure was 50 mm. Hg. At this time the entire chamber was optically clear, but later a clouding of the aqueous appeared which was diagnosed as probably due to cholesterol. The cholesterol content of the serum was 144 mgr. per cent.

The typical microscopical appearances in an eye which was excised for glaucoma of long standing, and was stained with sudan III, were as follows: The cornea showed a fatty infiltration of the parenchyma; the sclerotic was fat-free, except in its inner part. The choroid was fat-free, except for fine fatty bodies in some of the cells of the pigment epithelium. The retina was fat-free. The vitreous contained some resemblances of cholesterol crystals, which, however, were not typical. The iris showed an area loaded with fat arranged with a ring-shaped distribution. The anterior chamber was fat-free.

The author's experimental work dealt with the feeding of rabbits on a high cholesterol diet. The eyes were then wounded, and allowed to heal up with infection. Twelve days later the animals were killed, when examination showed a wide-spread xanthomatosis of the iris and ciliary body.

W. S. DUKE-ELDER.

(3) **Sobhy, M. (Cairo).—Cavernous angioma of the orbit and its ablation.** *Bull. Ophthal. Soc., Egypt*, 1927.

(3) In **Sobhy's** case the eye was pushed forward, down, and out. All movements were free. No tumour or resistance was felt around the globe. The proptosis was not reducible. There was no pulsation, murmur, or glandular enlargement. The pupil was semidilated and sluggish. The optic disc was swollen : the vision was only 4/60, partly due to the corneal opacity, the result of old trachoma. Wassermann negative. Radiography gave no help. Blood-count showed no increase of white blood corpuscles.

The operation was carried out, under general anaesthesia, through an angular incision at the outer angle of the orbit. A tumour was felt behind the globe to the outer side of the optic nerve. The tumour was shelled out easily; it measured 22 by 17 mm. It was found on microscopical examination to be an angioma.

A. F. MACCALLAN.

(4) **Peters.—Detachment of Descemet's membrane due to operative opening of the anterior chamber. (Ablösung der Membrana Descemeti durch Operationen, die den Vorderkammer eröffnen.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXX, S. 87, 1928.

(4) **Peters** reports two cases of detachment of Descemet's membrane after operation. The detached portion of the membrane in each case rolled itself up in the form of a cylinder, and remained quite clear. The first case was of a woman, aged 86 years, who was suffering from a chronic glaucoma, and on whom a cyclodialysis had been done. Twelve days later slit-lamp investigation showed a detachment of Descemet's membrane over a small area, surrounded by pigment changes in the cornea. The second case was of a woman, aged 87 years, who had absolute glaucoma. In her case an iridectomy was performed which was followed by the same sequela. This operative complication is extremely rare, but has been noted before.

W. S. DUKE-ELDER.

(5) **Villard, H. (Montpellier).—The simulation of bilateral amaurosis. (La simulation de l'amaurose bilatérale.)** *La Clin. Ophtal.*, February, 1927.

(5) **Villard** relates in full all the circumstances attending the examination of a case of simulated bilateral amaurosis in a soldier, who, after all, was never convicted, in spite of the most trying tests that can well be imagined. Nevertheless, it was afterwards found that when discharged from military service he was living happily and seeing perfectly, and even making a good living, in his native village. The details of the tests made in this extraordinary case in order to convict the man of fraud must be read in the original.

ERNEST THOMSON.

(6) **Loddoni, G. (Turin).—Case of hysterical manifestations after ocular injury. (Cas de syndrome oculaire hystérique.)** *Ann. d'Ocul.*, T. CLXVI, p. 357, 1928.

(6) **Loddoni** reports in detail the occurrence in a girl, aged 29 years, of conjunctival haemorrhage after a slight injury by fragments of metal. The vision of the affected eye was found on one occasion to be 6/6. Later, amblyopia and amaurosis developed in association with mydriasis, attacks of nystagmus and intermittent squint. The matter is discussed, and references are made to other cases in the literature. A bibliography is appended.

HUMPHREY NEAME.

(7) **Picard, P. (Thionville), and Dreyfus, P. A. (Strasburg).—Compression of the thorax followed by immediate and permanent blindness. (Compression du thorax suivie de cécité immédiate et definitive.)** *Arch. d'Ophtal.*, June, 1928.

(7) **Pickard** and **Dreyfus**, in the case now published, add one to the small number hitherto reported. They refer to seven examples in the literature, the earliest of which was published in 1906 by Béal. In all the instances loss of or severe damage to sight ensued, with the ophthalmoscopic appearances of optic atrophy. Picard and Dreyfus's case presented some unusual characters, or at least some which had not been noted in former cases.

The man, aged 37 years, was crushed by a travelling crane. On admission to hospital immediately afterwards, he exhibited the characteristic ecchymotic mask, with extensive haemorrhages of face and neck. He was quite conscious. There were several fractures of ribs and an acromian dislocation of the left clavicle.

Extensive ecchymosis of the eyelids, a high degree of chemosis, and subconjunctival haemorrhage occupying the whole of the ocular conjunctiva were noted. There was also noticeable exophthalmos, especially on the right side. The pupils were wide and immobile. The ocular media were clear, and the fundi quite normal, except for a small patch of old choroiditis in the left eye.

The man had no perception of even concentrated light, and stated that his sight failed suddenly about two seconds after he was struck. Signs of atrophic changes in the optic disc appeared about ten or twelve days after the accident; in four weeks the discs were completely blanched with sharp edges and narrowed arteries.

The authors discuss the various hypotheses which have been offered to explain the causation of the blindness in these cases: they incline to the hypothesis of haemorrhage in the substance of the nerve, followed by sclerosing degeneration. In none of the reported cases has there been post-mortem evidence of the lesions resulting from this type of injury.

J. B. LAWFORD.

(8) **Roggenbau and Wetthauer. — The permeability of the refractive media of the eye of the ox to long light waves. (Ueber die Durchlässigkeit der brechenden Augenmedien für langwelliges Licht nach Untersuchungen am Rindsauges.)** *Klin. Monatsbl. f. Augenheilk.*, Bd. LXXIX, S. 456, 1928.

(8) **Roggenbau** and **Wetthauer** have investigated the permeability of the ocular media to infra-red rays. They used the eyes of dead oxen, and, as a source of light, a Nernst lamp (temperature 2,261°). The light from this was conveyed through a slit aperture by a system of lenses and mirrors, and was dispersed by a prism. Measurements were taken by a micro-radiometer. They investigated the spectral region from 656 A.U. to 3,000 A.U. They recall the work of Vogt who measured the transmissibility of the human eye to infra-red rays by a galvanometer and a thermopile. It will be remembered that this investigator found that the cornea absorbed 20 to 25 per cent., the aqueous 20 to 30 per cent., the lens 30 per cent., and the whole of the ocular media 60 per cent. of the incident radiation. He also stated that rays from 800 to 1,500 A.U. penetrated the media, while those above 1,500 A.U. were absorbed.

The present investigators find that the permeability does not stop at 1,500 A.U. but goes on to 2,500 or 3,000 A.U. They find that the cornea has a maximum permeability between 900 and 1,000, that the aqueous and vitreous have maxima in the visible spectrum, while the lens has a maximum between 700 and 800

A.U., that is, in the region between the visibles and the infra-red. The permeability of all the media falls in the region of 1,400 to 1,500 A.U. to a first minimum, below which the permeability of the vitreous falls to *nil*. This phenomenon explains the findings of Vogt. Thereafter, however, the permeability of the other media has a second rise between 1,600 and 1,700 A.U. and reaches a second minimum between 2,000 and 2,100 A.U. After this a small third maximum is reached about 2,300 A.U.; thereafter the permeability gradually falls away. The importance of this investigation is the demonstration that a considerably higher percentage of radiant energy in the long waves of the spectrum falls upon and is absorbed by the lens. This tissue shows a maximum absorption of between 1,000 and 1,200 A.U. and again between 1,500 and 1,900 A.U. The authors' results are interesting in that they corroborate almost entirely the researches of Hartridge and Hill (1916) on the transmissibility and absorption of infra-red by the media of the eye, a research which appears to have been overlooked.

W. S. DUKE-ELDER.

(9) **Lacarrère, J. L. (Corunna). — Ocular biomicroscopy. A double slit-lamp. (Biomicroscopie oculaire. Lampe à double fente.)** *Rév. gén. d'Ophtal.*, April, 1927.

(9) It would be impossible to do justice to **Lacarrère's** article in an abstract, since the understanding of it depends almost wholly on the illustrations. The author describes a diaphragm—which can be fitted to the usual Zeiss instrument—containing two slits of peculiar shape and arrangement which greatly facilitate examination of the cornea, but which, it is specially stated, are not concerned with, and, so far as the author can see as yet, are not advantageous in examination of other parts of the eye.

ERNEST THOMSON.

(10) **Roy, J. N.—Electric dazzling. Medico-legal considerations. (L'éblouissement électrique. Quelques considérations médico-légales.)** *Ann. d'Ocul.*, T. CLXV, p. 433, 1928.

(10) **Roy** reports in detail the development of optic atrophy, with reduction of vision of the right eye of a workman to perception of light, as the result of exposure to the light produced by electric welding. For several months after the exposure, the vision of the affected eye was reported by various observers to be 1/10. About eight months after the exposure Roy found a retinal scar to the temporal side of the optic disc, a lesion of the macula and

optic atrophy. Careful investigations were made to exclude other causes of optic atrophy. He noted a deficiency of information on this matter in ophthalmic text books, but found reports of several cases in the literature (Brière, Rohmer, Panas, Knies, Terrien, etc.) in which optic atrophy and loss of vision had followed exposure to lightning or other electric discharge at close quarters. Apart from transient signs and symptoms in the skin and conjunctiva, the left eye of Roy's patient remained healthy. This is explained by presuming that the patient had turned his head to the left so that the left pupil was protected by the nose from direct exposure. A large bibliography is appended.

HUMPHREY NEAME.

(11) **Spindler, Henry (Lille).—The influence of the loss of one eye on the visual function and the consequences of this from the standpoint of capacity for work. (Influence de la perte d'un oeil sur la fonction visuelle et ses conséquences au point de vue de la capacité de travail)** *La Clin. Ophtal.*, January, 1928.

(11) **Spindler** combats the opinion that has been held by some writers that when one eye has been lost, and when time has brought about cerebral re-education the working capacity is not really seriously diminished, and, *a fortiori*, when there is merely diminution of visual acuity of one eye. These writers say that the only loss is that of one-sixth of the visual field. Spindler reminds us of the anatomy of the oculo-cerebral connections with special reference to the macular bundle, and makes the point he wishes to insist on in the following words: "When one eye has been lost the two cerebral macular visual centres, right and left, continue to be excited by the luminous impressions transmitted to one centre by the remaining portion of the macular bundle, for example the direct bundle, and to the other visual centre by the other portion of the macular bundle of the remaining eye, that is by the indirect bundle. The two visual centres thus continue to receive luminous impressions from the internal and external, superior and inferior sectors of the retina of the remaining eye, but, in so far as intensity is concerned, they receive one half only of the luminous impressions, since there is only one retinal surface instead of two to receive and transmit impressions. The fact that the two visual centres continue to be excited accounts for re-education in the estimation of distance and in the sense of relief."

The author gives an explanation as to how this comes about, but as the reviewer is unable to follow the explanation exactly in a foreign language it will suffice to say that in the author's view the

two cerebral centres are, after a time, able to build up perspective and relief sensations out of the messages received from the two halves of the remaining retina. In any case (and one is inclined to think this expression reveals a doubt as to the correctness of his theory of re-education), says the author, there is a diminution of the energy of vision (*énergie de la vision*). He compares the conditions to those of an under-exposed photographic plate compared with the same plate exposed for a longer time, and, similarly, points out how in one's every-day practice one finds that a normal individual sees the photometric scale more brightly illuminated with two eyes than with one eye, and how when, in an ametrope, each eye has been given by glasses an acuity X, both eyes together have an acuity X plus. The sum total of the author's calculations of incapacity is that a workman who has lost one eye has lost one-sixth of his field of vision, an amount, which he says, is agreed and is equivalent to an incapacity of 16.6 per cent., *plus* a loss of 8.4 per cent., due to " diminution of visual energy," the total " invalidity " due to the absence of one eye being thus 25 per cent.

ERNEST THOMSON.

(12) **O'Brien, C. S. (Iowa).—Local anaesthesia in ophthalmic surgery.** *Jl. Amer. Med. Assoc.*, January 27, 1928.

(12) This is a useful description of the means of securing anaesthesia in ophthalmic surgery by instillation, infiltration, and nerve-block, using cocaine, novocaine and various other substitutes for cocaine. For cataract extraction **O'Brien** recommends instillation of cocaine four times, and then a subconjunctival injection of equal parts of a 2 per cent. solution of novocaine and 1/1,000 adrenaline solution. He then blocks the facial nerve in the following manner: The point of injection is just anterior to the tragus of the ear, below the posterior portion of the zygomatic process and directly over the condyloid process of the mandible. Going straight inwards with a short needle one strikes the bony condyloid process at a depth of about 1 cm. As soon as the bone is felt with the needle about 2 c.c. of a 2 per cent. solution of novocaine is gradually injected in withdrawal. (Nerve-blocking is more usually carried out at the present day by the use of a much more dilute solution of novocaine, say 0.25 per cent., in much larger quantity, 10 or 15 c.c.—Reviewer.)

O'Brien says: " Except in cases of enucleation or evisceration or in an eye that is blind, it is usually not wise to inject deep into the orbit for fear of injuring some of the structures lying therein."

A. F. MACCALLAN.

(13) **Galal-Aboul-Seoud (Cairo).—Four weeks ophthalmic work in Mecca.** *Bull. Ophthal. Soc., Egypt*, 1927.

(13) **Galal-Aboul-Seoud** is an experienced ophthalmic surgeon who had an opportunity to spend four weeks in 1926 in the Islamic Holy City of Mecca, the capital of the Hejaz. Here there is a general dispensary which was founded in 1820 by the Albanian Governor of Egypt, Mohammed Ali, the ancestor of the present King of Egypt. The work was carried on at the dispensary during the month of February, the total number of patients examined being 200. The operations included 18 for cataract, 9 for glaucoma, 48 for trichiasis or entropion, and 40 for trachoma.

The author was particularly struck by the absence of trachoma among the inhabitants of Mecca, their eyelids being perfectly smooth. Such evidences of trachoma as he saw were among the people of Nejd, who are now occupying the city. Nejd is an emirate of central Arabia extending eastwards from the Hejaz to the Persian Gulf. It consists mainly of a plateau where rain is prevalent, and where consequently there is cultivation. The climate is cooler than in the Hejaz and there are frequent high winds and sand storms.

The inhabitants of Mecca, on the other hand, live in a valley, very warm in winter, and in the summer unbearably hot. There is practically no rain, and absolutely no cultivation. The extremely limited water supply is brought in pipes for more than 20 miles. The author suggests tentatively that this immunity of the inhabitants of Mecca to trachoma may possibly depend on the meteorological conditions. He considers that there is no racial distinction between the inhabitants of Mecca and their neighbours the inhabitants of Nejd. He is unable to offer an entirely satisfactory explanation of the immunity of the inhabitants of Mecca and the Hejaz generally, though they have been visited annually for centuries by thousands of pilgrims from trachoma-stricken countries.

A. F. MACCALLAN.

BOOK NOTICES

Recent Advances in Neurology. By W. Russell Brain, D.M. (Oxon.) and E. B. Strauss, B.M., B.Ch.(Oxon.) With 38 illustrations. London: J. and A. Churchill. 1929. Price, 12s. 6d.

This is an excellent book which should be read by all ophthalmologists. There are astonishingly few pages in it which have not some lesson for them. The resemblances of the mechanism of nutrition of the brain and of the eye—secretion, and circulation of cerebro-spinal and aqueous fluids, normal and pathological variation of intracranial and intraocular pressures, etc.—have a distinct bearing not only on the ocular manifestations of intracranial tumours, but also on the pathology of glaucoma: in the latter respect the chapter on the treatment of increased intracranial pressure by means of hypertonic solutions is specially interesting. Ophthalmic surgeons will naturally turn to the chapters on intracranial tumours—especially those involving defects of the visual fields—and the pituitary gland and hypothalamus for authoritative information as to recent knowledge, and they will not be disappointed. Scattered elsewhere they will find chapters on encephalitis lethargica, Schilder's disease, dystrophia myotonica, etc., where the very prominent ocular signs must be duly regarded and appraised by both ophthalmologists and neurologists. They are urged, however, also to read other chapters which do not at first suggest an immediate ophthalmic interest. One of the most important of these is that on neurotropic viruses, which contains an excellent account of the pathogenesis and pathology of herpes (febrilis and zoster), poliomyelitis, etc. It forms an excellent introduction to the study of virus diseases, the interest of which is not limited to acute inflammations but spreads to all the problems of immunity and malignant growths.

Having studied these chapters the ophthalmologist will doubtless be drawn on to read those on posture and tonus, on conditioned reflexes, on sleep, and on sensation, which, if they have little direct bearing on the practice of ophthalmology, are full of scientific interest.

Lehre von den Gesichtsempfindungen. By Franz Hillebrand. Edited by Dr. Franziska Hillebrand. Pp. 205. Vienna: Julius Springer. 1929. Price, 14 Reichsmark.

Our earliest experience of "things" in the outer world is derived from the bombardment of our bodies with stimuli. These

stimuli fall upon an organism whose make-up is the result of aeons of evolution. In due course, through speech, we are bombarded by the concepts which form the tradition of our race and which are the outcome of experience. This tradition is embedded in the words which make up language. It has no scientific validity as an expression of ultimate truth other than that derived from the gropings after truth as the explanation of the phenomena of the experience of our parents and fellows and their ancestors. Thus it comes about that we " see objects," and say that " the moon is bright," " the grass is green " and " an orange is—orange!" In these latter days most of us go to school, and in due course are introduced to the mysteries of physics. We then learn that Newton, by means of a prism, split up white light into a spectrum, and that Huygens and Thomas Young said it consisted of waves, and eventually we learnt that red light had a wave-length of 600—700 $\mu\mu$ and green light a wave-length somewhere about 500 $\mu\mu$. We were then taught that it was not the moon which was bright or the grass green, but that these qualities were bound up with the light which emanated from these objects. And that suffices for most people. But some of us went on to study physiology and especially that part of physiology called neurology. We then learnt that light of wave-length 700 was not always red, nor that of 500 always green; they might both of them be white; and when they were mixed together in suitable quantities they were always very nearly white. So we were then taught that the colour was not inherent in the light, but in our brains, which ultimately received the impulses which were set up in our retinae by the light waves. A few of the physiologists went a step further and realised that the colours and so on were not even inherent in the physiological impulses, but that somehow or other these gave rise to sensations and perceptions which were facts of consciousness or mind. And it occurred to them to start at the purely conscious end of the story and more or less ignore all these complicated physical and physiological processes.

The greatest of these psychological physiologists—at any rate in this particular domain—was Hering. Looking at colour, for example, from this point of view he practically said, " So far as my consciousness is concerned, apart from light and dark and the intermediate ' greys,' there are only four colours, red, yellow, green and blue: all the others are psychologically mixtures of these." Following out this train of thought all the phenomena of colour vision assumed a quite novel aspect as compared with looking at them from the other point of view. Both points of view led to hypotheses and speculations which were very difficult to reconcile with each other. The physical point of view and its theories appealed naturally most to physicists, and indeed were much more easily understood by most people because they nearly all knew

some physics, whereas very few of them knew much physiology and practically none of them any psychology, at any rate any psychology founded on biological foundations. Nevertheless the physiologists were much attracted by Hering's views, largely because he was himself a great physiologist and because he stated his theory in physiological terms. Unfortunately he published a large amount of his work in journals like *Lotus*, which were not readily accessible. It has long been desirable that Hering's researches and views should be made accessible in an exhaustive and authenticated manner.

The book which has given rise to these lucubrations to a very large extent fulfils this requirement. Hillebrand was one of the staunchest of Hering's disciples and probably reflects his master's views better than any one else. He unfortunately died without gathering together the scattered threads of Hering's argument. But he left ample materials and fortunately one who was capable of dealing with them adequately. His widow, Frau Dr. Franziska Hillebrand, has done the work well, adding in places some useful explanatory and elaborating notes.

The book is divided into two parts, dealing respectively with the light sense (96 pages) and the space sense (72 pages): a final section deals with the old—indeed now somewhat antiquated—discussion on nativism and empiricism. In the first part the dependence of colour sensation on the stimulus leads to a description of the methods and laws of colour mixture. To those familiar with Helmholtz and the physical school the description is far from complete and leaves much to be desired; but it is illuminating in revealing some of the weaknesses of the Hering school. The dependence of colour sensation on the excitability of the receptive organ reveals the strength of the Hering views in dealing with adaptation and contrast, and its weakness in dealing with colour blindness. The section on colour theories should be read by all interested in the subject, since it represents the arguments of the perfect Heringite.

It is probable, in the opinion of the reviewer, that Hering's work on spatial localisation will outlast that on colour vision. It is well treated in somewhat compressed form in this book. It is at least as well treated, and indeed, more fully and comprehensively elsewhere, as, for example, in Hofmann's *Die Lehre vom Raumsinn des Auges*. Nevertheless, it is instructive to read the unadulterated creed of the Hering school.

The book is a worthy monument, not only to the author and his wife, but also to Hering, their great source of inspiration.

Photographs of the Fundus Oculi. By ARTHUR J. BEDELL, M.D. An Atlas containing 95 Plates, 324 Single Pictures and 272 Stereoscopic Photographs. Philadelphia: F. A. Davis Company. 1929. Price, 25 dollars.

The title page gives an adequate indication of the nature of this book. The photographs, 1⅜ inch in diameter, are probably as good as can be obtained at present. In our opinion they are a mere *tour de force*, and of little value for teaching purposes. We frankly fail to understand the point of view of the author, who writes that, as compared with coloured drawings: "By the use of photographs it is possible for physicians to get a clearer idea of the disease with a better conception of the outline of the blood-vessels, the optic nerve and the innumerable retinal and choroidal variations. The absence of colour in the picture tends to emphasise these details."

Considering the high illumination necessary to photograph the fundus we doubt if the method is legitimate for normal eyes, *a fortiori* for those suffering from inflammatory diseases of the retina, choroid, and optic nerve

Surgical Pathology. By CECIL P. G. WAKELEY, F.R.C.S. and St. J. D. BUXTON, M.B., B.S., F.R.C.S. Pp. 864, 392 illustrations. Bristol: John Wright & Sons, Ltd. Price, 45s.

This is a book written primarily for students of general surgery with the object of helping them with their clinical work. It is admirably illustrated and contains excellent accounts of the various surgical conditions. The first portion comprises general pathology; inflammation, wounds, gangrene, tumours, etc., while the latter part of the book is concerned with the special pathology of the different organs and regions in the body.

With regard to ophthalmological subjects, there is a short description of glioma of the retina, of uveal growths and of inflammatory and neoplastic diseases of the orbit and lids. One would not expect the descriptions to be as full as in a text book on ophthalmic pathology, but in the reviewer's opinion, it would have been advantageous to have included a note of the macroscopic appearances in choroidal sarcoma and in glioma of the retina since the authors' wish is "to encourage the student to read these pages while he has specimens beside him." Also in the description of the microscopic appearances in glioma, although degenerative changes are mentioned no reference is made to mantling, a phenomenon which by itself is usually sufficient to enable the student to identify a section of this tumour. There is a useful account of pituitary growths in which the symptoms are grouped under the

headings: (1) Those due to alteration of secretion. (2) Those due to local pressure on surrounding structures. (3) Those due to increase of intracranial pressure. It is a pity that the words " optic neuritis " although followed by (" choked disc ") should still occur in the description of the third group of symptoms. It might also have been worth while emphasising the occasional association of papilloedema on one side with optic atrophy on the other, as this, when present, is such a valuable sign in the diagnosis of pituitary growths.

Surgical Pathology is an admirable book for the general surgeon. It is admittedly not written for the specialist, and the latter, particularly in ophthalmology, has no cause for dissatisfaction as he is already amply catered for in the well-known works on the Pathology of the Eye. We venture to suggest, however, that an occasional excursion into the realms of pathology outside the orbit cannot but be good for him, and that for this purpose he could not do better than turn to the pages of the book under review.

Index of Symptomatology. By VARIOUS WRITERS. Pp. 687. Bristol: John Wright and Sons, Ltd. London: Simpkin, Marshall, Ltd. 1928. Price, 42s.

This is a comprehensive volume covering all branches of medicine and surgery. It contains the work of 26 separate contributors. To review it is therefore a task rather akin to reviewing say the Encyclopaedia Britannica. The book should, however, serve as a useful vade-mecum to those engaged in general practice and much care and thought must have gone to the writing of its contents, since the object of this has been to give a clear and reasonably full description of the clinical manifestations of each disease, without dwelling unduly on minor complications or variations.

Ophthalmology has perforce had to be content with the relatively small space of 20 pages, but in this space, P. G. Doyne has managed to give many of the important symptoms of common eye diseases, and he has presented them in an attractive manner, not in any way suggestive of a synopsis or cram book. Numerous omissions have had to be made and it would be easy to criticise the author's contribution on this score, but we would rather admire the skill he has shown in making such a comprehensive survey of ophthalmic symptomatology in so small a space. In his own particular sections of the book, the ophthalmic surgeon will probably find little that he did not know before, but let him stray into other parts and his pride will soon be humbled, also he will soon realise how far away his narrow path of ophthalmology may have led him from the broad highway of general medicine and surgery and their allied special subjects.

NOTES

Honour

AMONG the New Year's honours we are pleased to note the name of Lt.-Col. R. E. Wright, I.M.S., of Madras, who receives the C.I.E.

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Appointments

MR. A. SOURASKY, F.R.C.S., has been appointed Ophthalmic Surgeon to Out-patients, Hampstead General and North-West London Hospital.

MR. H. E. QUICK, M.B., F.R.C.S., has been appointed additional Specialist Medical Referee to take ophthalmic cases in the County Courts of Aberayron, Aberystwith, Cardigan, Carmarthen, Lampeter, Llandilo, Fawr and Ammonford, Llandovery, Llanelly, Narberth and Haverfordwest, Neath and Port Talbot, Newcastle-in-Emlyn, Pembroke (Circuit 31).

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Oxford Ophthalmological Congress

THE provisional programme of the Nineteenth Annual Meeting of the Oxford Ophthalmological Congress is as follows:—

July 4, discussion on "Diseases of the lacrimal apparatus," introduced from the ophthalmological standpoint by Professor J. Meller (Vienna), and from the rhinological aspect by Dr. J. S. Fraser (Edinburgh).

July 5, Doyne Memorial Lecture by Miss Ida Mann, the subject being "Some observations on the vascularisation of the vertebrate eye." In the afternoon there will be a discussion on the "Ocular manifestations found in fractures of the skull." This will be opened from the aspect of the general surgeon by Mr. Hugh Cairns (London), and from the ophthalmological side by Mr. C. Goulden (London).

In addition to the foregoing, the following papers and demonstrations have been promised: Dr. Maddox, Demonstration of the cheiroscope and frame fitting by photography; Mr. Thomson Henderson, Further evidence of the absence of healing of the cut surface of the iris after iridectomy; Mr. Ridley, An unusual complication of Hodgkin's disease; Dr. Samuels, Necrosis of the iris; Mr. Hughes, The structure of the vitreous; Mr. Young, Treatment

of conical cornea: Effects of hyperaemia in the treatment of incipient cataract.

Applications for membership should be made to the Hon. Secretary, C. G. Russ Wood, 12, St. John's Hill, Shrewsbury.

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La Société française d'Ophtalmologie

THE Forty-second Congress of the Société française d'Ophtalmologie will take place at Paris, in the Faculté de Médecine, on Monday, May 13, and following days. The opening paper of the customary symposium will be read by Dr. Aubaret, of Marseilles, on "The aetiology and treatment of blepharitis." An exhibition of optical and surgical instruments will be shown. Visits to laboratories and hospitals will be made. M. Maurichéau-Beaupré will conduct an excursion to visit the Trianons on Tuesday, May 14; and the customary banquet will be held at Versailles the same evening. Further information may be obtained from the Secretary, Dr. René Onfray, 6 avenue de la Motte Picquet, Paris VII.

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A Library of Medical History

THE St. Louis Medical Society has been presented with a valuable collection of books dealing with medical history by Dr. Moores Ball. The collection comprises 1,000 volumes dating from 1502—1928; 1,500 medical portraits, and several thousand pamphlets, reprints and newspaper cuttings.

FUTURE ARRANGEMENTS

1929

April 11-13.—Ophthalmological Society of the United Kingdom. (Annual Congress), London.

May 13-16.—Société française d'Ophtalmologie. (Annual Congress), Paris.

June 14.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting).

July 4-5.—Oxford Ophthalmological Congress, at Oxford.

September 5-13.—International Congress of Ophthalmology at Amsterdam.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *January, 1929.*

DAILY, LOUIS, and DAILY, RAY K. Diagnosis and treatment of dacryocystitis.
BERENS, CONRAD, and LOSEY, RAY R. Vaccine therapy in diseases of the eye.
COURTNEY, ROBERT H. The use of lens protein tests before cataract operations.
RODIN, FRANK H., and HALL, ERNEST M. Nevus of the bulbar conjunctiva.
GRADLE, HARRY S. An unusual cyst of the forehead and orbit.
PASCAL, JOSEPH I. A sensitive test for equality of accommodation.
LANGDON, H. MAXWELL. Cataract wound infected with facultative anaerobic streptococcus.
MCKELLAR, J. H. Epiphora due to relaxation.

February, 1929.

CREBBIN, ALEXANDER R. Persistent pupillary membrane and congenital ectopia lentis.
MORTON, HOWARD MCI. Intracapsular extraction without iridectomy.
RING, G. ORAM. A recent visit to the Barcelona clinic of Dr. Ignacio Barraquer.
EBER, CARL T. The conjunctival bridge in cataract operations.
LOY, A. W. A study of the factors concerned in depth perception.
GOLDENBURG, MICHAEL. A case of malignant lymphoma.
BROWN, ALBERT L. Subconjunctival injections of atropin and adrenalin in iridocyclitis.

Archives of Ophthalmology. *January, 1929.*

CUSHING, HARVEY, and EISENHARDT, LOUISE Meningiomas arising from the tuberculum sellae with the syndrome of primary optic atrophy and bitemporal field defects combined with a normal sella turcica in a middle-aged person. *(To be continued).*
WILMER, W. H. Diseases of the eye in old age.
VERHOEFF, F. H. Histologic observations in a case of localised tuberculous chorioretinitis.
GRAEFE, ALBRECHT VON. Iridectomy in glaucoma: a study of the glaucomatous process.
DONNELL, N. R. Ulceration of the sclera.

The Journal of Ophthalmology, Otology and Laryngology.

December, 1928.

ATKINSON, T. D. The problem of the secondary cataract.
ROWLAND, WILLIAM D. Foreign body in orbit.

Annales d'Oculistique. *January, 1929.*

REDSLOB and REISS. On the experimental variations of the intraocular tension. Its relation to chemico-physical properties of the vitreous.
BIDAULT. Contribution to the anatomical and physiological study of the retinal circulation in old age.

CARLO. The monocular perception of depth and its consequences.
CATTANEO. Some observations on the mechanism of accommodation.
ADAMANTIADIS. A case of malignant hyperplastic conjunctivitis.

Société Belge d'Ophtalmologie.

COPPEZ. A case of melanosarcoma of the choroid with multiple metastases.
Epithelioma of the limbus followed for 18 years.
Stereoscopic photographs of the anterior part of the eye.
Photograph of the retina with the aid of Nordenson's chamber.
BRANDÈS. A rare occupational accident.
DANIS. Cyst of the bulbar conjunctiva.
WEEKERS and HUBIN. Xanthelasma, generalised xanthoma, hemeralopia and chronic jaundice.
WIBO. Abnormal functional synergism of lateral movement and closure of the lids.
KLEEFELD. Treatment of orbital cellulitis by osmotic drainage.
Local insulin therapy of severe pneumococcal ulcers.
Ocular biomicroscopy.
MICHAUX. Strange case of oscillating oculo-motor paralysis.
VAN LINT. Traumatic retinal haemorrhage with abolition of vision.

Archiv für Ophthalmologie. *January, 1929.*

CASCIO. Research on the refraction in the human eye with obliquely incident light.
POOS and SANTORI. The effects on pupil movement and accommodation of sympathetic nerve poisons in cases of relatively raised blood-aqueous threshold.
NIITANI. Experimental research into pupil innervation.
ENGELKING. "Ground" and "figure" and their meaning in the colour vision of anomalous trichromats.
RICHTER. On the presence of colour in the tapetum and the difference between tapetum cellulosum and fibrosum in the eyes of domestic mammals.
FRISCH and PILLAT. On the question of tuberculous irido-cyclitis and the aetiology of irido-cyclitis of unknown origin.
ROHRSCHNEIDER. Experimental researches into the changes in the tissue of the normal eye exposed to X-rays. Part i. and ii.
KYRIELEIS. Clinical, anatomical and experimental researches on papilloedema.

Klinische Monatsblätter für Augenheilkunde. *January, 1929.*

FRANCESCHETTI and GUGGENHEIM. The erythrocyte separation time and its meaning in ophthalmology.
REHSTEINER. Notes on our knowledge of capsule glaucoma (glaucoma capsulocuticulare).
MINDER. On the visibility with the slit-lamp of changes in the corneal nerves in leprosy.
HAUSSEN. On the question of albuminuric retinitis.
SCHULTE. On striate epithelial keratitis.
PETERS. A malformation of the inner canthus.
KILNECKER. On a case of bilateral so-called lentiglobus anterior.

LOWENSTEIN. On the clinical picture of anaphylactic ophthalmia with reference to the pathogenesis of interstitial keratitis.

CATTANEO. Notes on our knowledge of the zonular lamella.

THIEL. Staphylococcal metastases in the region of the anterior ciliary vessels.

SATTLER. Severe connective tissue haemorrhage in the new born.

KLAUBER. Foreign body in the anterior chamber as a reason for complications after cataract extraction.

ROSENTHAL. Natural union of the anterior chamber with the subconjunctival tissue and high hypotony.

SCHÖPFER. Traumatic implantation cyst of the ciliary body.

ZIRM. Further notes of the forward and outward section of the sclera in glaucoma.

OPPENHEIMER. A riddle.

Annali di Ottalmologia e Clinica Oculistica.

January, 1929.

OVIO. Influence of black on vision.

BASSO. On compensating glaucoma in myopes.

SCULLICA. The behaviour of the endothelial reticulum in some experimental pathological processes in the eye.

LODDONI. Copper cataract.

SCOTTI. On a case of retinitis punctata albescens.

ANTONIBON. Quantitative researches on the absorptive power of the cornea.

BARLETTA. Hypertension in the differential diagnosis between glioma and pseudo-glioma.

Archivio di Ottalmologia. *December, 1928.*

ANGELUCCI. The phenomenon of sublimation in the art of Michaelangelo, Wagner and Berlioz (finis).

CAVANIGLIA. Therapy of chronic dacryocystitis.

TIRELLI. On ocular symptoms in Vaquez's disease.

Anales de la Sociedad Mexicana de Oftalmologia y Oto-rino-laringologia. *July and August, 1928.*

ALONSO. Pterygium on the coast of the Gulf of Mexico.

VÉLEZ. Application of photography to the study and examination of ocular disease.

Revista Oto-Neuro-Oftalmologica y Cirurgia Neurologica.

February, 1929.

HARTMANN. Retro-Gasserian neurotomy and its ocular effects.

GOERLITZ. Reciditive exophthalmos from frontal empyema.